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Important Books Received

Technic of oral radiography. By Clarence O. Simpson, M.D., D.D.S., F.A.C.D. 1926: pp. 207; 6¾ x 4 inches; 165 illustrations; $5.00. C. V. Mosby Co., St. Louis.

Orthodontics: an historical review of its origin and evolution. *Two volumes.* By Bernhard Wolf Weinberger, D.D.S., Librarian of First District Dental Society of the State of New York, and Advisory Librarian, Dental Department, New York Academy of Medicine, New York City. 1926: pp. 1011; 7¼ x 4¾ in.; 599 illustrations; $25.00 (*two volumes*). C. V. Mosby Co., St. Louis.

American text-book of operative dentistry. By Marcus L. Ward, D.D.S., D.D.Sc., F.A.C.D., Editor; Professor of Dental Metallurgy and Crown and Bridge Work, and Dean, College of Dental Surgery, University of Michigan. *Sixth edition.* 1926: pp. 818; 7¼ x 4½ in.; 783 illustrations; $10.00. Lea and Febiger, Philadelphia and New York.

A textbook of exodontia—exodontia, oral surgery, and anesthesia. Leo Winter, D.D.S., Professor of Oral Surgery, College of Dentistry, New York University. 1927: pp. 364; 7¼ x 4¾ in.; 329 illustrations; $7.50. C. V. Mosby Co., St. Louis.

Harelip and cleft palate (cheiloschisis, uranoschisis, and staphyloschisis). By Matthew N. Federspiel, B.Sc., D.D.S., M.D., F.A.C.S., Professor of Oral Surgery, Marquette University. 1927: pp. 200; 6¾ x 4 in.; 156 illustrations; $5.00. C. V. Mosby Co., St. Louis.

A text-book of bacteriology for dental students. By Arthur Bulleid, L.R.C.P. (London), M.R.C.S., L.D.S. (England), Assistant Dental Surgeon and Assistant Bacteriologist, Guy's Hospital, London. 1927: pp. 219; 6½ x 4 in.; 60 illustrations; $4.50. C. V. Mosby Co., St. Louis.

Lectures [6] on the biologic aspects of colloid and physiologic chemistry. A series of lectures given at the Mayo Foundation and the Universities of Minnesota, Iowa, Washington (St. Louis), and the Des Moines Academy of Medicine, Iowa. By six investigators in the field. Introduction by Louis B. Wilson, M.D., Director of the Mayo Foundation for Medical Education and Research. 1927: pp. 244; 6¼ x 3¾ in.; 85 illustrations; $2.50. W. B. Saunders Company, Philadelphia apd London.

American illustrated medical dictionary. By W. A. Newman Dorland, A.M., M.D., F.A.C.S., member of the Committee on Nomenclature and Classification of Diseases, American Medical Association, and Editor of the "American Pocket Medical Dictionary;" with the collaboration of E. C. L. Miller, M.D., Professor of Bacteriology and Biochemistry, Medical College of Virginia. *Fourteenth edition.* 1927: pp. 1388; 8¼ x 4¾ in.; 319 illustrations; flexible binding—plain, $7.00; thumb index, $7.50. W. B. Saunders Company, Philadelphia and London.

Dental education in the United States and Canada. By William J. Gies, M.S., Ph.D., Sc.D., LL.D., F.A.C.D., Professor of Biological Chemistry, Columbia University; Executive officer of the Board of Editors, *Journal of Dental Research;* with a preface by Henry S. Pritchett, Ph.D., Sc.D., LL.D., Litt.D., President of the Carnegie Foundation for the Advancement of Teaching. 1926 (issued in 1927): pp. xxii + 692; 7½ x 5 in.; one illustration; distributed free of charge on application, by letter or in person (the volume is "Bulletin No. Nineteen"). Carnegie Foundation for the Advancement of Teaching, 522 Fifth Avenue, New York City.

Physical chemistry and biophysics, for students of biology and medicine. By Matthew Steel, Ph.D., Professor of Biological Chemistry, Long Island College Hospital. 1928: pp. 372; 7¼ x 4¾ in.; 38 illustrations; $4.00. John Wiley and Sons, Inc., New York City.

Fundamental principles of a systematic diagnosis of dental anomalies, with an explanation of a new classification based upon gnathostatic methods of investigation. By Paul W. Simon, Head of the Department of Orthodontics, Dental Institute, University of Berlin. *Translated* by B. E. Lischer, D.M.D., F.A.C.D., Non-resident Lecturer on Orthodontics, Dental School, University of Michigan. 1926: pp. 378; 6½ x 4 in.; 189 illustrations; $5.00. Stratford Company, Boston, Mass.

The practice of periodontia. By Sidney Sorrin, D.D.S., Chief of Periodontia Clinic, and Samuel C. Miller, D.D.S., Instructor, Periodontia Clinic, College of Dentistry, New York University. 1928: pp. 160; 6 x 3¾ in.; 122 illustrations; $3.50. MacMillan Company, New York.

The romance of the atom. By Benjamin Harrow, Ph.D., Associate in Biological Chemistry, Columbia University. 1927: pp. 162; 5¼ x 3¼ in.; 7 illustrations; $1.50. Boni and Liveright, New York.

The nature of matter, gravitation, and light. By Albert P. Mathews, Ph.D., Professor of Biochemistry, University of Cincinnati. 1927: pp. 222; 6¾ x 4 in.; 7 illustrations; $7.00. William Wood and Co., New York City.

The religion called behaviorism. By Louis Berman, M.D., Associate in Biological Chemistry, Columbia University. 1927: pp. 153; 5¼ x 3¼ in.; no illustrations; $1.75. Boni and Liveright, New York.

Dental state board questions and answers. By R. Max Goepp, M.D., Professor of Principles and Practice of Medicine and of Clinical Medicine, Woman's Medical College of Pennsylvania, Philadelphia. *Fourth edition.* 1928: pp. 463; 7¼ x 4¾ in.; no illustrations; $4.50. W. B. Saunders Co., Philadelphia and London.

American universities and colleges. By David A. Robertson, Assistant Director, American Council on Education, Washington, D. C. 1928: pp. 884; 7¼ x 5¼ in.; no illustrations; $2.50. Charles Scribner's Sons, New York and London.

An introduction to organic chemistry. By Alexander Lowy, Ph.D., Professor of Organic Chemistry, University of Pittsburgh; and Benjamin Harrow, Ph.D., Associate in Biological Chemistry, Columbia University. *Second edition.* 1928: pp. 407; 7 x 4 in.; 22 illustrations; $3.00. John Wiley and Sons, Inc., New York.

A text-book of general bacteriology. By Edwin O. Jordan, Ph.D., Professor of Bacteriology, University of Chicago and Rush Medical College, Chicago, Ill. *Ninth edition.* 1928: pp. 778; 7¼ x 4¾ in.; 191 illustrations; $6.00. W. B. Saunders Co., Philadelphia and London.

Laboratory manual of physiological chemistry. By Meyer Bodansky, Associate Professor of Biological Chemistry, and Marion S. Fay, Adjunct Professor of Biological Chemistry, School of Medicine, University of Texas. 1928: pp. 234; 7¼ x 4¾ in.; nine illustrations; $2.00. John Wiley and Sons, Inc., New York City.

DENTAL RESEARCH IN THE UNITED KINGDOM[1]

SIR WALTER FLETCHER, K.B.E., F.R.S.

London, England

I think it is a great privilege to be allowed to be with you here today, and to join both in your annual celebration and in the special event of this year, the opening of your new research laboratory. With all your other guests, and I am sure I may speak here on their behalf, I would offer our congratulations to the whole School upon the successful year of work of which we have just had so good an account. We would offer our warmest good wishes to the students now beginning work here, and to those who, on completing their course, are now passing on to professional life. Of these, we have greeted some who have just received their well-deserved prizes. Those who have not won prizes today will no doubt reflect that as all experience shows, though success no doubt in general breeds success, the order of merit of today will by no means necessarily be in the same order of merit ten, twenty, or thirty years hence. Hopeful as that reflection will be to them, it may also be by no means without its helpfulness to the prize men of today, whose success now gives such good augury for the future.

The special event of today is the first opening of the new research laboratory, which has been built here by aid of a grant from the United Kingdom Board. The equipment and upkeep have been provided in chief part by the generosity of Mrs. Hale, who dedicates her gift to the memory of her husband, John Hampton Hale, formerly Chairman of the Hospital. This laboratory will add greatly to the resources of the School, and we shall wish earnestly that the work done in it may both win renown here and serve the good of mankind everywhere.

[1]Address delivered at the prize distribution and the opening of the John Hampton Hale Research Laboratory, at the Royal Dental Hospital of London School of Dental Surgery, on October 4, 1927.

This new laboratory is undoubtedly a valuable addition to the School; but the significance of our ceremony today goes far beyond that. The laboratory is an outward and visible expression, perhaps the first which the public has had an opportunity of noting, of a new and a very striking movement in the dental profession. I doubt very much whether the general public have any conception of the new policy by which the members of the dental profession are themselves directly supporting and promoting both education and research in the art and in the sciences of dentistry. Under the provisions made by the United Kingdom Dental Board, every practitioner registered since 1921 pays not only a registration fee, but also an annual fee for retention on the Register. As the Register grows, the body of contributors to the fund will grow and eventually the whole profession will be included. The fund already amounts to about £40,000 a year, and this is spent under the management by the Board in promoting education and research. Dental education is promoted by assistance given to able students needing it, by the provision of "refresher" courses for practitioners seeking them in special directions, and in other ways. Dental research is aided by grants made to workers under approved schemes.

The public ought not to fail to notice and to appreciate this most honorable and enlightened policy of the dental profession, at present almost unique in its kind. It will be seen that the dental practitioners are directly contributing with goodwill to a Fund for promoting the two great movements that in a narrow view might seem to be most damaging to their immediate self-interest: that of education, which is designed to bring into the competitive field against them the ablest and best equipped rivals; and second, that of research, of which the deliberate aim is to prevent dental surgeons so far as possible from having any work to do at all. Could there be a more notable example of public-spirited altruism?

Both these great movements, thus supported by the profession itself—that towards the improvement and accessibility of professional education, and that towards the advance of knowledge by research—are movements made in the best interests of the profession, viewed narrowly and selfishly; but they will inevitably promote the honor of the profession that identifies itself in this way with the common inter-

est, and will deserve and receive general admiration as they become increasingly known.

Now that the lead has been given by the dental profession, we may all wish that admiration by other professions of their movement may presently stimulate imitation. Supposing members of the medical profession each paid a small annual contribution in recognition of the privileges given them by registration, and that the fund so produced was devoted in the high spirit of altruism to the interest of the public by being used to promote improved and better sustained medical education and in gaining improved medical knowledge through research. Do you realize that if each doctor in the United Kingdom contributed what each dentist now contributes, there would be a fund of no less than a quarter of a million sterling annually for medical education, whether undergraduate or post-graduate, and for medical research? A fund of that kind might aid notably in the solution of some long-standing but pressing national problems of post-graduate education in medicine.

Important and hopeful as is the new policy framed and financed for the profession by the Dental Board, we cannot properly appraise its value in this time of sowing. The harvest time is still to come. This is most obviously true in the field of dental research, and yet here we can already see even in these early days some beginnings of an actual harvest and the rich promise of more to come.

In framing their research policy the Dental Board have recognized that, however precise and limited are many of the special practical aims to which dental research is directed, the scientific work by which alone these aims can be realized must have complete liberty of range, if it is to be new and progressive, and must be free to bring into contribution any suitable parts of the great primary sciences. In the important technical field of reparative dentistry they have entered into coöperation with the Department of Scientific and Industrial Research, and, for study of their special problems in mechanics, metallurgy, and refractory materials, have thus taken into alliance the whole system of scientific effort throughout the country with which that department is in touch. I believe that definite and valuable gains of a practical kind have already been the result even at this early date.

I am able to speak with more direct knowledge of the similar co-operative alliance which has been effected, between the Dental Board and the Medical Research Council, in respect of research in dentistry upon that preventive and curative side of it which is so important a branch of medicine. There are few problems of preventive medicine of more significance to the race, and not least to the people of our nation, at present, than that of the proper development and preservation of the teeth. Every modern gain in our knowledge only serves to emphasize in some new way the scientific and medical interest of this set of problems. The Council I serve have recognized this from their beginning, and so far as their means allowed have lost no opportunity of assisting work in what is in many respects a peculiarly difficult field. They greatly welcomed the coöperation which the Dental Board offered them four years ago, and now the two bodies join together in financing the schemes of research recommended and generally supervised by an investigation committee upon which laboratory workers in bacteriology, biochemistry, and nutritional physiology sit side by side with clinical and anatomical observers under the chairmanship of that distinguished member of your Staff here, Mr. Norman Bennett. My Council believe, and they hope the Dental Board also are satisfied, that by this partnership economy is well served and scientific efficiency increased. It will be our hope that the new laboratory we are to inaugurate today will play its own distinguished part in this coöperative scheme of investigation.

Who can doubt the value and the pressing urgency of this research work from the point of view of the citizen and the nation? Dental imperfections and dental disease in this country bring directly and immediately a colossal and dismal volume of discomfort, bad health, inefficiency, pain and ugliness. Indirectly, disorders of the teeth, besides their immediate and direct effects, may contribute in various degrees to specific diseases of the body. It is now almost a commonplace to say that the improvement of the teeth of the nation is one of the most urgent of the great outstanding problems of preventive medicine.

Yet while we recognize the urgency of the research work by which alone in our present ignorance we can hope to find the solution of this national problem, it is useless and perhaps worse than useless to

demand or expect quick and easy results from the investigations. Money is necessary to get freedom and opportunity for the right workers, and money invested in research rarely fails in the long run to give a high return of interest and often at an almost fabulous rate. But you cannot, by merely putting money down, either secure quick results or indeed command any at all. New natural knowledge cannot be bought, it is the product of the right man in the right place and of his unfettered labor. As Bishop Hall, of Norwich, said truly three hundred years ago: "There was never good thing easily come by . . . . God sells knowledge by Sweat." There is another warning also to be given. The endowment of research not uncommonly leads to a good deal of research after endowment. There are always those, whether trained or untrained, who are ready to undertake investigation in any direction without definite clue or clear objective. The true limit of a research program is fixed not by the money at disposal but by the number of men or women both fit and available for the enterprise, and they will always be scarce.

Will you forgive me if I remind you, too, that we must not only be patient in waiting for results, but catholic in our scientific spirit? With a definite utilitarian goal before us, as here, that of preventing dental disorders, there is a common danger of expecting success from the frontal attack, and of neglecting to observe and foster the progress of those who, perhaps in some quite different field of work, are reaching a point from which a flank attack may gain the whole position we are seeking. Nature's clues rarely lie in the straight line, and the key of some great advance in dental knowledge may be lying implicit in studies that seem now to be quite alien. To those who can help us by some other road upon the flank, we must be more encouraging than the Irishman who, when asked by a belated traveller the way to Roscommon, said: "Bedad and it's a long way. If you're going to Roscommon, you shouldn't be starting from here."

It would be impossible here and now to give any fair account in detail of what the Investigation Committee supported by the Dental Board have actually accomplished already. That is easily to be found elsewhere. I would ask leave only to offer my own reasons for thinking that the scientific workers they have assisted have already brought the problems of dental disease into a new atmosphere of

hopefulness and interest. The study of the attacks made on the teeth by particular agents of disease had proved before, and seems still to prove, to be a relatively barren study. What has been needed, and what the Committee have been giving their chief energies to obtaining, has been better knowledge, not so much of dental disease as of dental health; not so much of the specific diseases that may attack the teeth, as of the conditions upon which their natural resistance to disease depends. Recent work in this country has drawn increasing attention to the exquisite elaboration of the minute architecture of the teeth, not only as it is built up when the tooth is first constructed for its long years of hard service, but also in the processes of its repair against later damage. Evidence has been accumulating that if this architecture be perfect, as it should normally be, the attacks of disease are prevented or minimized. It has also become known that ordinary inspection by the eye as made hitherto does not reveal this architectural health of the tooth. The tooth may seem to be normal in color and shape, and yet the microscope may show its intimate construction to be faulty. These considerations seem likely to clear up, on further study, many puzzling problems of decay in teeth apparently, but only apparently, sound.

But recent work has been doing much more than this. It has been increasingly bringing these dental problems into line with those of the general physiology of the body and the science of its nutrition. Clear experimental evidence has already been obtained that the normal architecture of the teeth, like that of the bones and other organs of the body, depends upon the right supply and balance of particular parts of the diet during their growth. It seems, moreover, that, whether from the very intricacy of their wonderful structure or whether for other reasons, the teeth in their formation are even more sensitive to nutritional conditions of given kinds than other parts of the body. Certainly we all know how often the human body, splendidly formed and functionally fit in every other way, is marred only by faulty teeth. It seems not unlikely that dental practitioners in the future may have new and wider service to give, by aiding their colleagues in other medical fields to use rightly the sensitive index to normality, that is to health, which the teeth in their formation and repair seem destined to provide.

I said that the progress made in these directions was full both of hope and of interest. It is full of hope because we seem to be in sight of new powers altogether, in the light of new knowledge; new powers, that is, of determining the structure and the functional health of the teeth by proper adjustment of diet at the right time. It is full of interest because at every step in this work problems of deep significance in biological science are being raised and illuminated. It has special interest, too, because the advances made here are all tending to bring preventive dentistry into new and closer relations to the general body of medical science. We are only seeing the dawn now of nutritional knowledge, and the light is still so dim that it hardly serves even yet to show us our ignorance. The hints we are fast gaining, however, seem to make it no rash speculation to say that the medicine of the future lies in the lap of nutritional science.

However this may be, we must press forward in hope and with patience. Solomon said in his wisdom: "Hardly do we guess aright at things that are upon earth, and with labor do we find the things that are before us." Here and today in your new laboratory we dedicate a fresh enterprise in the long quest for knowledge; we shall pray that it may serve well the highest interests of your profession, and bear its due part in winning more knowledge for the dignity and interest of mankind, and more power for his help in this and other nations.

AN M.D. DEGREE FIVE YEARS AFTER HIGH SCHOOL*

WILBURT C. DAVISON, M.D.

Duke University School of Medicine, Durham, N. C.

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Every one interested in medical education regrets that 26 years is the average age at which the M.D. degree is received;[1] yet many hesitate to reduce the time required for fear of lowering the present standards. Three years can be taken from this average age and placed on the credit side of a man's life by admitting to medical school younger, intelligent students after two years of college work and by utilizing the summer quarters so that the length of the medical course can be reduced to three years. These ideas are not new,[2] but Duke University, unhampered by tradition and previous condition of servitude, will have an opportunity to try the experiment when the School of Medicine opens in 1930 provided, of course, that the medical faculty which has not been appointed approves the plan.

* Read before the Annual Congress on Medical Education, Medical Licensure, and Hospitals, Chicago, February 6, 1928. Reprinted from the *Journal of the American Medical Association* (1928, xc, p. 1812; June 2), by courtesy of the editor and with the consent of the author.

[1] Rappleye, W. C.: Second report of the Commission on Medical Education, January, 1928.

[2] Harper, W. R.: Educational plan, in Goodspeed, T. W.: History of the University of Chicago; University of Chicago Press, p. 140. Wilbur, R. L.: *Proceedings of the Annual Congress on Medical Education, Medical Licensure, and Hospitals*, Chicago, February, 1926, pp. 20–21. Bass, C. C.: Discussion of the quarterly basis for the Tulane School of Medicine, May 25, 1926; Is it advisable for medical schools of the South to adopt the four quarter system? Read before the Section on Medical Education, Southern Medical Association, Atlanta, Ga., November 15, 1926. Graves, M. L.: The four-quarter system and the fifth or intern year, *Southern Medical Journal*, xx, p. 963 (December) 1927. Colwell, N. P.: The relation of the quarter system to the medical licensing, *Federation Bulletin*, xiii, p. 115, 1927.

I. AN ENTRANCE REQUIREMENT OF INTELLIGENCE AND CHARACTER PLUS TWO YEARS OF COLLEGE WORK

Thirty-five years ago the entrance requirement of a college degree, which was instituted by the Johns Hopkins University School of Medicine, was undoubtedly advisable, for the high schools and preparatory schools of that time were not all that they might have been, and four years of college work was necessary to obtain a basic education. However, a comparison of the average high school curriculum of today with that of most of the colleges of 1893 is, except for the courses dealing solely with the classics, often in favor of the high school. In other words, the entrance requirement for a college degree in 1928 is comparable to that for a college degree plus four years of postgraduate study in 1893. Is it not time to give the student the benefit of this advance in education and, since now at the age of 18 years he can obtain a preliminary education as good as that formerly received at the age of 22, to admit him to medical school without a college degree? I do not believe, however, that a high school certificate should be accepted for admission to medical school, even though it may now be equivalent to the average college diploma of the early nineties, for the amount of premedical science—chemistry, biology and physics—which a medical student should know today has greatly increased, and two years of college work is necessary in order that the student may obtain this preparation in science.

It is true that 81 per cent of the class A medical schools in this country require only two years of college work for admission,[3] but it is invariably the rule that preference is given to the candidate who has attended college for three or four years. It is the proud boast of most medical deans that their students are practically all college graduates. Why? Probably because of the supposition that the quantity of hours spent in college has some relation to the quality of the student's intelligence and that to enforce the two-year requirement would lower the standards. Some schools give a "grade bonus" to college graduates, which not infrequently gives a college graduate who has done mediocre work an advantage over a much better student

[3] Summary of admission requirements of Class A Medical Schools: *Bulletin of the Association of American Medical Colleges*, ii, p. 333, 1927.

who has applied for admission on the basis of a two- or three-year preparation.[1]

The careful selection of younger, intelligent students, instead of lowering the present standard of physicians, will probably raise it. Medical education consists of three periods: the premedical, or high school and college; the medical, or medical school; and the postmedical, or hospital internship. All three are important, but under existing conditions the third is often reduced because the medical graduate of 26 or 27 years usually feels that one year is all he can afford to spend in a hospital. Would he be a better physician if he had a longer hospital apprenticeship? The answer is obvious. Two-thirds of a group of successful practitioners recently studied[1] had more than one year of internship. Mastery of any subject is impossible in medical school. The student usually acquires a certain number of facts, methods of study, a knowledge of how to approach medical problems and of where and how to obtain more information; but unless the graduate digests and assimilates this mass of education by learning its practical application in a hospital, it is soon forgotten and wasted. Many outstanding medical students have become mediocre practitioners because of this lack of postgraduate training, while three years of hospital work has frequently transformed an average student into a brilliant physician. One need only recall the great mental as well as physical improvement shown by physicians who have developed tuberculosis and have had, against their will, as it were, to obtain from one to three years of additional hospital training at Saranac Lake or other sanatoriums.

I believe that the most important way by which additional hospital training can be made more universally possible is through a reduction in the age of the medical graduate. The premedical and medical periods can and should be shortened. The younger applicants should be given the preference, and the entrance requirements should be intelligence and character, plus two years of college work, including two years each of English and chemistry and one year each of biology, physics and mathematics. This preliminary training should fit the keener and more ambitious students for entrance into medical school. Perhaps for the slower student, four years of college work is necessary, but that type is not under discussion here. The objection might be

raised that it is too difficult to select this bright student; but the experience at the Johns Hopkins University School of Medicine has demonstrated that this selection is not difficult. There, for the past three years, only those college graduates have been admitted who had intelligence and character, qualifications which have been adjudged from personal interviews and confidential reports from their science teachers rather than from the quantity of their preparation, and the annual number of their failures has been reduced from more than 15 per cent to less than 2 per cent.[4] Our endeavor would be to admit this type of student after his second year in college rather than after he had been graduated. It cannot be denied that, as a result of the sifting process, the chances are that those who survive four years of college work are likely to be a more highly selected group than those who have had only two. However, I feel that if the committee on selection makes the necessary effort, the exceptional students can be recognized. If this experiment does not succeed the entrance requirements can, of course, be changed.

It is realized that the greatest weakness in our present educational system is the time wasted in grammar schools and high schools, and that if the brighter students were allowed to progress in accordance with their capabilities, instead of plodding in lock-step with the average mass, they could profitably enter college at the age of 15 years. President Lowell[5] says: "In our effort to push all children through the educational mill, have we not unwittingly sacrificed the more intelligent to their own damage?" However, this selective type of early education seems to be too expensive and too difficult politically for tax support. The attitude of one superintendent of schools is expressed as follows:[6] "The preparation of boys and girls for college is a mere detail in the broad program of a great high school. Our objective is not to train a chosen few for higher education, but to prepare all our students for American conditions of life." Furthermore, since 82 per cent of physicians do not decide to study

[4] Davison, W. C.: The selection of medical students, *Southern Medical Journal*, xx, p. 955 (December), 1927.

[5] Lowell, A. L.: Address read before the National Education Association, Boston, February 27, 1928.

[6] Boynton, F. D.: Address read before the National Education Association, Boston, February 27, 1928.

medicine until the age of 15 years,[1] this valuable time in grammar and high school is already lost.

In practically all universities, a keen student can obtain the necessary training in English, chemistry, biology, physics and mathematics in the first two years. However, in some institutions those in charge of these departments do not feel that they can adequately teach their subjects in this time. It is undoubtedly true that mastery of any one of these subjects cannot be acquired in two years any more than mastery of any subject in medicine is possible in the time allotted to it in the medical curriculum. Compromises are necessary between those who wish their students to obtain a foundation in science so that they can become well rounded physicians and those who wish to train their students to become expert chemists or physicists, and who look on the problem from the point of view of the gross number of hours allotted to their own subjects. All branches of science have grown so vigorously and have put forth so many offshoots that the question now, to quote Berglund,[7] is: "What to teach and what to skip."

The importance of modern languages is frequently emphasized, and an additional year or more in college is recommended for this preparation. Thirty years ago a reading knowledge of German and French was necessary for advancement in medicine, but at present practically all of the foreign medical literature is quickly translated and abstracted, and readily available to those who cannot read the original articles. It is true that the latter are consulted by a small percentage of physicians, but it would seem unnecessary to require every student to spend two or three years in the study of German and French. Those who need this knowledge will acquire it anyway, while those who have it and do not use it will quickly forget it. At the Johns Hopkins University School of Medicine every entering student can read, in some fashion or other, German and French. However, four years later, at the time of graduation, less than 10 per cent of the students can read either language at all, as a result of disuse atrophy. Either these languages should be discarded as entrance

[7] Berglund, H.: What to teach and what to skip, *Proceedings of the Association of American Medical Colleges (38th Annual Meeting)*, Montreal, Canada, October 24–26, 1927.

requirements or else the medical schools should resort to grammar school methods and hold frequent recitations in German and French.

Four years in college are also held to be desirable for their cultural value. However, culture seems to be acquired by some persons without college training and never obtained by others in spite of numerous humanistic courses. As a matter of fact, the majority of those who have culture have gained it at home, or from associations and environment early in life. Others, however, with the same advantages remain unsaved. Stewart Roberts[8] has recently commented on the superiority of the culture of British interns. The average British medical student acquires his cultural background at home and at school, and rarely studies cultural subjects at a university. At Oxford, for example, all of his time is spent in biology, physics, chemistry, anatomy, physiology, and biochemistry.

It is frequently stated that the four years in college are especially valuable for those who later are to do scientific work in medicine. However, it would seem that for this group the requirement of four years of college work, which is enforced either by rule or by preference, is really a handicap. There are many investigators who have spent the major portion of their four years in college in the study of biology and who now find in their chosen field that a training in chemistry would have been preferable. If these men were two years younger, they could return for the additional basic training in chemistry.

It is sometimes stated that a medical school cannot obtain a sufficient number of these outstandingly intelligent students to fill the class. Consequently, medical schools which must fill their classes to capacity hesitate to maintain a rigid entrance standard based on the students' intelligence and character, and prefer to select students on the basis of the quantity of their preparation, for it is easier to fill a medical school class with college graduates than it is with students who are keen and ambitious. It is probably true that the Duke University School of Medicine will not be able to fill its classes to capacity with this type of student but, if not, the classes will not be filled to capacity, for the late Mr. James B. Duke, in his deed of gift,[9] requested

[8] Roberts, Stewart, in a discussion on "Changes suggested for the medical curriculum," *Southern Medical Journal* (to be published).

[9] Jenkins, J. W.: James B. Duke, master builder, p. 294. George H. Doran Company, New York; 1927.

"that great care and discrimination be exercised in admitting as students only those whose previous record shows a character, determination and application evincing a wholesome and real ambition for life."

Many committees on admission grant the reasonableness of these contentions, but give the applicant with a college degree the preference on the grounds that the four years in college have certainly not done the student any harm and that most students will spend the additional time in college with very little urging. I am not at all sure that the additional time in college is without harm for, in colleges in which the gifted student is not given special advantages,[10] so little effort is required for the keener students to maintain the college standards which are set for the average mass that habits of academic indolence frequently develop.

One argument advanced in favor of the collegiate degree for entrance is that since the college graduate is older he therefore is more mature, more responsible, more serious, and more industrious. It is probably quite true that students 22 years of age work harder than those two or three years younger; but their doing so is more of a necessity than a virtue: they must work harder because they usually learn more slowly. As a matter of fact, if the admission committee performed its function properly, it could select applicants who had these desirable qualities in spite of being only 19 or 20 years of age.

A college degree has also been recommended in order that the present age at which a physician enters practice may not be reduced. I do not agree that a physician or surgeon need be over 28 years of age[1] before he can assume the responsibilities of practice, for there are too many outstanding examples to the contrary in this country and abroad; but, even granting this, I believe that two additional years in a hospital or laboratory after graduation will be more valuable training than two additional years in college before entering medical school. I am making a plea for younger medical graduates rather than for younger practitioners. Let the same age for engaging in practice be maintained if that is desirable, but send a better trained man into practice.

There is also a tradition that students who have had four years

[10] Few, W. P.: The gifted student and the American college. President's Report to the Trustees of Duke University, 1928.

of college work make better records in medical school than those who have had two or three years. But do these students maintain this record because of their additional training or because they were keener and more ambitious anyway? On the other hand, to quote Manning,[11] "We have had in our classes year after year students with two, three, and four years of college and I doubt that any one except the dean knows who of the students have had two, or three, or four years of college. In spite of the fact that the majority of students entering medical school today are college graduates, the percentage of failures during the first two years in medical school is practically as high as it was fifteen years ago, when the majority of medical students had only one or two years of college preparation." It is interesting that more than half of the group of successful practitioners recently studied had two years or less of college work before entering medical school.[1]

One of the greatest difficulties in the way of persuading the keener type of students that an entrance requirement of two years of college is sufficient is the great desire on the part of most of them to have a college degree and to graduate with their class. This dilemma is solved in some institutions by granting a B.S. or an A.B. degree after two years of college and two years of medical school. This pernicious system of granting double rewards for the same work has debased the value of these degrees. A better policy would be to grant a B.S. degree to medical students who, in their free time in medical school, had done creditable, extra work in one or another department and had written a thesis. In this way the degree would represent additional work accomplished, and would also serve as an incentive and stimulus to acquire more medical knowledge than was required for the M.D. degree. It is expected that this plan will be adopted at the Duke University School of Medicine.

This whole problem of two or four years of college work for entrance to medical school naturally raises the question of the origin and rationale of the four year college course. Apparently the four-year curriculum dates back to the statute adopted in February, 1252, which specified that students of the English nation at the University

[11] Manning, I. H., in a discussion of a paper by Davison (footnote 4).

of Paris should study at least four years for the bachelor's degree.[12] In the British and European universities there is no such set time period. Their students, after fulfilling certain minimal requirements, take the examinations for their degrees as soon as they feel that they are in a position to pass them. At Oxford, "candidates for the degree of Bachelor of Arts are required to pass three examinations, and to keep nine Terms (three academical years) by residence."[13] Some students wait six years before qualifying, while those with senior standing can do so in two years.

TABLE 1

Calendar for curriculum for four quarters of eleven weeks each (1927–1928)

Autumn quarter, *October 3 to December 23*	
Thanksgiving vacation	*November 23 to 28*
Christmas vacation	*December 23 to January 2*
Winter quarter, *January 2 to March 17*	
Winter vacation	*March 17 to 26*
Spring quarter, *March 26 to June 9*	
Spring vacation	*June 9 to 18*
Summer quarter, *June 18 to August 31*	
Summer vacation	*August 31 to October 1*

There is a movement on foot at present to break away from the four-year rule. In the academic department of the Johns Hopkins University, students who have creditably completed the first two years of college, either there or elsewhere, will be admitted to untrammeled study in any academic department. In various cities throughout the country junior colleges have been created in connection with the high schools at which a student may obtain the equivalent of the first two years at a university. At one well known university some of the alumni, realizing that many of the students are more interested in obtaining a college degree for social and business purposes than in acquiring an education, have proposed that the course be shortened to two years. In other words, junior colleges of freshmen and sopho-

[12] Wilkins, E. H.: The reasons why four has become the accustomed number of years in the extension of academic and professional curricula, *Journal of Dental Research*, vii, p. 513, 1927; *Bulletin of the American Association of College Registrars*, ii, no. 4, 1927.

[13] University of Oxford, General information concerning admission, residence, etc., Oxford at the Clarendon Press, p. 7, July, 1926.

mores and senior colleges of juniors and seniors may become part of our educational structure.

II. A CURRICULUM OF THREE CALENDAR YEARS OF FORTY-FOUR WEEKS EACH

Another year can be saved by condensing the four medical school years of thirty-three weeks each into three years of forty-four weeks each (*Table 1*). The advantages to the medical student are obvious. He will be one year younger at graduation and will have an additional

TABLE 2

Curriculum based on four quarters of eleven weeks (429 hours) each

	Hours		
First Year			
Autumn quarter (1st)			
Anatomy (including histology)	429		
Free time		0	
Total			429
Winter quarter (2d)			
Physiology	160		
Biochemistry	144		
Free time		125	
Total			429
Spring quarter (3d)			
Pharmacology	56		
Bacteriology	88		
Psychobiology	16		
Free time		269	
Total			429
Summer quarter (4th)			
Free time		429	
Total			429
Second Year			
Autumn quarter (5th)			
Pathology	288		
Free time		141	
Total			429
Winter quarter (6th)			
Clinical microscopy	96		
Medical psychology	16		
Physical diagnosis	160		
Preclinical examinations	39		
Free time		118	
Total			429

Spring quarter (7th)*			
Medicine (junior) (including pediatrics)	296		
Free time		133	
Total			429
Summer quarter (8th)*			
Surgery (junior)	144		
Obstetrics (including gynecology)	108		
Free time		177	
Total			429
Third Year			
Autumn quarter (9th)*			
Specialties (junior)	112		
Free time		317	
Total			429
Winter quarter (10th)*			
Medicine (senior; including pediatrics)	312		
Free time		117	
Total			429
Spring quarter (11th)*			
Surgery (senior)	256		
Free time		173	
Total			429
Summer quarter (12th)*			
Final clinical examination	39		
Free time		390	
Total			429
Summary:			
Total number of hours of required instruction	2,759		
Total number of hours of free time	(54%)	2,389	
Total number of hours in curriculum		(46%)	5,148 (100%)

* The clinical instruction should be repeated each quarter in order to utilize all the clinical material and to have small groups of students. Consequently, students may vary the order of the seventh, eighth and ninth quarters; and also the order of the tenth, eleventh and twelfth quarters.

year for hospital or other training; he will be better prepared, for he will not have lost a fortnight or a month in October of each year getting back into the intellectual stride, which had been his in the preceding June, and he will see the clinical material peculiar to the summer months.

The disadvantages to the medical student have been overemphasized. For example, it is said that he needs the four months

of vacation. However, practically all medical students at the present time work during their summer holidays; 50 per cent study preclinical or clinical subjects in some place or other; and if this time were spent under supervision at a medical school, the character of the work would be improved and much valuable time saved. The 50 per cent who earn money during the summer vacations could probably earn

TABLE 3

Changes from the four quarter schedule necessary for students who do not attend the summer quarters

First Year:	
Autumn quarter (1st)	No change from schedule
Winter quarter (2d)	No change from schedule
Spring quarter (3d)	No change from schedule
Second Year:	
Autumn quarter (4th)	Same as fifth quarter in schedule
Winter quarter (5th)	Same as sixth quarter in schedule except that the preclinical examinations are postponed to the end of the spring quarter and the amount of free time increased correspondingly to 157 hours
Spring quarter (6th)	Same as fourth quarter except that the preclinical examinations are held and the amount of free time correspondingly decreased to 390 hours
Third Year:	
Autumn quarter (7th)	Same as seventh quarter in schedule*
Winter quarter (8th)	Same as eighth quarter in schedule*
Spring quarter (9th)	Same as ninth quarter in schedule*
Fourth Year:	
Autumn quarter (10th)	Same as tenth quarter in schedule*
Winter quarter (11th)	Same as eleventh quarter in schedule*
Spring quarter (12th)	Same as twelfth quarter in schedule*

* The clinical instruction should be repeated each quarter in order to utilize all of the clinical material and to have small groups of students. Consequently, students may vary the order of the seventh, eighth and ninth quarters; and also the order of the tenth, eleventh and twelfth quarters.

more in the year which they will save under the four-quarter system than in the three periods of four months each. The establishment of loan funds, rather than scholarships, will do much to solve this phase of the problem. It has been said that a medical student cannot physically endure the work of forty-four weeks a year. If so, he probably should enter some less arduous occupation, for as a physician

he undoubtedly will have to work as hard or harder for longer than forty-four weeks a year. The total of eight weeks of vacation, which every student will have under the four-quarter system (*Table 1*), should be sufficient for any one. The argument that the summer climate of Durham or of any other city would be injurious to medical students would be vigorously answered by weather statistics of chambers of commerce, and also by the realization that the major part of the population remains at work all summer and that practically all the physicians continue their practices. As a matter of fact, if any student prefers to study four calendar years of thirty-three weeks each he can do so under this flexible curriculum (*Table 3*). A certain number of students, either through illness or through financial difficulties, will probably be absent one or more quarters, but under this curriculum they can take up their work at the beginning of the next quarter and not lose a whole year as is necessary at present. This irregularity is in itself an advantage, for it will reduce the usual rigid lock-step succession of studies.

The fear has been expressed that, if the four-quarter system were adopted, it would be necessary to repeat numerous courses in order to accommodate both the students who desire the four-quarter system and those who attend only three quarters, and that a much larger number of teachers and a greater medical school budget would be required so that the staff could have the present academic vacations. It is probably true that under the rigid curricula used in most medical schools it would be necessary to repeat several required courses in order to accommodate the students who did not attend the summer quarters. However, if the present schedule of the Johns Hopkins University School of Medicine,[14] which has many advantages,[15] were adapted to the four-quarter system, as suggested in *Tables 1*, *2* and *3*, all the required preclinical courses would need to be given only once a year. The clinical courses would be repeated each quarter in order

[14] Johns Hopkins University School of Medicine, *Bulletin of the Association of American Medical Colleges*, ii, p. 341, 1927.

[15] Baker, L. H.: An experiment with the curriculum, *Proceedings of the Association of American Medical Colleges* (*38th Annual Meeting*), Montreal, Canada, October 24–26, 1927. Davison, W. C.: Liberalizing the curriculum, *Southern Medical Journal* (to be published).

to utilize all the clinical material and to have small groups of students. At first glance this would seem a hardship for the clinical staff; but, as this teaching is largely the demonstration of patients in the wards and outpatient department, it is very little if any more effort than attending these patients, which must be done anyway. As a matter of fact, medical students, after their first month of clinical training, are a great help in the care of patients and in the administration of the wards and the outpatient department. The vacations without students are usually more laborious than with them.

Approximately one-half of the time in this curriculum (*Table 2*) is free for elective work or anything else the student wishes to do. In the first year the greatest amount of free time is concentrated in the latter part of the year in order to give the student a more varied curriculum early in his course and thus enable him to choose elective courses from a wider range of subjects. The summer quarters of the first and third years have been left entirely free in the hope that many of the students will migrate to other medical schools in this country or abroad for elective work, a practice which should be encouraged. The establishment of the four-quarter system at Chicago, Tulane, and other universities, will greatly aid this exchange of students. For students who do not attend the summer quarters, the spring quarters of the second and fourth years are free for the same purpose (*Table 3*). The students who do not transfer temporarily to other medical schools may utilize their free time in elective courses in preclinical and clinical departments, may pursue independent work in any subject, or may do research work. The elective courses should be organized for small groups and repeated if necessary in one or more quarters.

Comprehensive group examinations in the preclinical subject would be given to the students as soon as they had completed six quarters, and in the clinical subjects at the end of the twelfth quarter. These examinations would be held at least twice each year to accommodate students who qualify for them at different times. They would demand a more comprehensive knowledge of medicine than could be obtained from the required courses in the schedule, and it would be necessary for the student to demonstrate that he had profitably utilized his free time.[15]

This curriculum is extremely flexible and, except for first year students, those who transfer from other medical schools can be admitted into any quarter for which their previous training has fitted them. For example, a student from a two-year medical school could enter the summer quarter in June and graduate in December of the following year, at the end of his twelfth quarter, or he could enter in October and graduate in March or June two years later, depending on whether he attended the intervening summer quarter. First year students would all be admitted at the same time, in October, in order to start their medical training with the required course in anatomy.

The size of the average staff and budget need not be increased in order to carry out this curriculum, for instead of all the members taking their vacations at the same time, they could arrange the teaching and decide among themselves the quarter during which each instructor would be away. It would be a great advantage to any member of the staff to be able to take his vacation during the months in which most medical schools in this country and abroad are in session. Furthermore, it would divide the responsibility of departmental administration, and give each first assistant an opportunity during the quarter in which his chief is away to gain the experience he needs and does not usually get. It is, of course, essential that the quality of the instruction during the summer quarter should be maintained at the highest level and not allowed to reach the depths of some summer courses.

There are numerous other advantages in the four-quarter system—the tremendous capital invested in medical schools will not be idle one-third of the time; the clinical material of the hospitals will be better utilized; the patients will receive better care, and the size of the individual classes can be reduced; or, if the present size of the classes is maintained, more students can be accommodated.

This three-year plan is apparently acceptable to the medical boards of licensure of thirty-one states[16] and it is hoped that the other seventeen will accept an intern year in lieu of the fourth calendar year of medical school.

[16] The quarter system in medical education: Council on Medical Education, pamphlet 159; Choice of a medical school, p. 20, November 1, 1927.

III. CONCLUSION

The present standards of physicians can be maintained and in fact probably improved if preference in admission is given to carefully selected younger students who have intelligence and character plus two years of college work, and if the M.D. degree is given after the completion of three medical school years of forty-four weeks each. The rigid selection of students is the essential factor in the success of this experiment.

DAVISON'S CONTRIBUTION TO AN UNDERSTANDING OF THE TWO-THREE-GRADUATE PLAN OF DENTAL EDUCATION

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Editorial Office, Journal of Dental Research

The preceding paper by Dean Davison of the School of Medicine of Duke University,[1] although relating directly to a study of medical education, is reprinted in this *Journal* because it has an important bearing on current problems in dental education. The paper will be particularly helpful to those dental teachers who, although desiring to facilitate the improvements assured by the two-three-graduate plan,[2] have not been able to persuade themselves that this plan will help dental schools to attain academic, scientific, and professional parity with medical schools, or that it will support the effort to make dentistry the service equivalent of an oral specialty of the practice of medicine.

It is a notable fact that although the Duke University School of Medicine will have exceptional financial support, its officers, in aiming to give the School the highest quality, have not been deluded into assuming that this is automatically attainable by erecting mechanical barriers to entrance, by prolonging residence in the institution, and by making the cost of a medical education excessive. It is evident, also, that the great gift upon which the School has been founded will be used with due regard for the economic welfare of the population of North Carolina.

The present writer, cordially appreciative of the great significance of Dean Davison's paper, notes a disagreement on a detail that may

[1] Davison: An M.D. degree five years after high school; *Journal of Dental Research*, 1928, viii, p. 473.

[2] Gies: A further discussion of some problems in dental education; *Proceedings of the American Association of Dental Schools* (First Annual Meeting), 1924, i, pp. 144–45. Also, *Bulletin on Dental Education in the United States and Canada;* No. 19 of the Carnegie Foundation for the Advancement of Teaching, 1926, pp. 202–206.

suitably be brought to the reader's attention. On page 480, in alluding to a desire of "the keener type of students . . . . to have a college [academic] degree and to graduate with their class," Dean Davison states that "this dilemma is solved in some institutions by granting a B.S. or an A.B. degree after two years of college and two years of medical school," and adds: "This pernicious system of granting double rewards for the same work has *debased the value of these degrees*" (italic not in the original). Many opinions recently expressive, however, of a different view regarding standardization of "the value of these degrees" might be quoted. Thus, President Lowell of Harvard University, in his Annual Report for 1924–25 (reprint, pp. 10–11), indicated as follows the feeling that these degrees are not, and need not be, uniformly equivalent in value:

"The experiment of eliminating the [academic] college in part is being widely tried in the case of men who intend to enter the professions of law or medicine, and to some extent other careers. It is done by the use of the 'combined degree,' whereby the last two years of college are spent in professional study, and are counted towards the degrees both of the college and of the professional school. . . . . Complaint has been made of the combined degree because for a single course of study, pursued through a couple of years, it gives a double credit toward two distinct degrees supposed to indicate different kinds of work. But it would seem to make little difference what degrees are named *if it is understood that they are not, and had better not be supposed to be, closely equivalent in all institutions* (italic not in the original). The object of the combined degree is to eliminate two years of the traditional American college education. In fact, this appears to be a common tendency in this country. In some of the western colleges—not connected with a university—a considerable part of the students leave before the end of the four years, often to take up professional studies in a university that provides a combined degree. For most American young men this gives as much college education as they can afford, and perhaps as much as under the circumstances is wise."

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DIAGNOSIS OF PERIODONTOCLASIA[1]

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I. INTRODUCTION

There is a tendency to hold civilization responsible for most of the ills that afflict us, and to assume that the wild animal in its native habitat is free from disease. Knowledge on this subject is of great importance. Review of the literature is convincing as to the occurrence of certain systemic diseases in prehistoric and primitive animals. MacCurdy (1) tells us that lesions similar to those produced in modern times by periostitis, necrosis, arthritis, and osteoma are found in the bones of dinosaurs, turtles, crocodiles, and other reptiles of the Mesozoic; that fossil animal remains from Tertiary deposits afford evidence of the existence of numerous diseases; and that fossil races of man were by no means free from many of the ills affecting the races of more recent times. To the question, "How and when did disease originate?", Dr. Saxton Pope (2) replies, "With life itself!" The conflict with bacteria, protozoa, and other pests has existed from the first biologic reaction at the beginning of organic evolution.

As to dental disease, in spite of the questionable reliability of some of the literature, the argument for the occurrence of dental caries in

[1] Presented at a session of the Sixth Annual Meeting of the International Association for Dental Research, Hotel Mayflower, Washington, D.C., March 25–28, 1928.

primitive animals appears cogent. Whether this is true of pyorrhea is still an open question. MacCurdy states that pyorrhea alveolaris dates from the paleozoic, and quotes Renault to the effect that caries is found in Permian fishes that lived twenty millions of years ago. In a personal communication, however, MacCurdy says that "Paleozoic pyorrhea has never been described in a satisfactory way, but there seems to be some evidence of it among the Permian fishes." Schultz reports cavities and alveolar abscesses[2] among monkeys living under perfectly normal conditions and shot in their native habitat.

The observations of Turner are of great interest in this connection. He found no evidence of dental sepsis in fish. Examination of scrapings of the mouth taken shortly after capture revealed no bacteria. In the large collection of skulls of wild carnivora (tigers, etc.), at the South Kensington Museum, he found no post-mortem evidence of dental sepsis other than abscess following a fractured tooth, except in an old hyena from Somaliland. The sepsis in this case followed gross injury to the gum by coarse food. In skulls of wild grass-eating animals he found no post-mortem evidence of dental sepsis. Films from the mouths of these animals showed large numbers of bacteria, but, with the exception of a mycotic infection, indicated no resultant disease. This mycotic infection was of the same nature as the localized pyorrhea of wild monkeys. Turner believes that alveolar disease of the domestic horse is due to spikes of chaffed hay speared into the gum, producing a localized not a generalized disease. Examination of skulls of wild monkeys, at the South Kensington Museum, showed 0.53 per cent of the teeth carious and 5.31 per cent of the skulls containing carious teeth. The skulls of seventy-two monkeys, which had died at the Lincoln Zoological Society Gardens, gave 2.001 per cent of teeth carious and 16.66 per cent containing carious teeth. Pyorrhea among wild monkeys, Turner believes, occurs only in its localized form. Bone may be found destroyed between two teeth, but the rest of the mouth remains sound. In the pockets, coarse spikes of vegetable origin are found, and the conclusion

[2] Alveolar abscesses in wild animals and in primitive man, resulting from excessive wear of the teeth leading to pulp exposure, or from fracture, are of course entirely different in significance from those which occur as sequelae to caries or pyorrhea. The former would be accidental injuries rather than true disease.

is inescapable that the disease was the result of trauma while eating. The skulls of captive monkeys show widespread alveolar changes comparable to those found in human beings. Tartar in small amounts is found in wild monkeys, but considerable quantities are present in captive monkeys.

On the basis of these data it would appear that *the assumption that dental caries is peculiar either to man or to domestication is misleading.* It follows as a corollary—and this is very important—*that the essential causes of this disease must be found in conditions common to widely variant forms of animal life, both feral and domestic, although, as will be shown later, domestication and civilization have profoundly increased the incidence of etiologic factors.* To what extent these statements apply to pyorrhea depends upon further and more careful observation. If convincing evidence is forthcoming that true pyorrhea does not occur in wild animals in their native states, then this disease will be restricted wholly to man (fossil and modern) and to domesticated animals, while dental caries will not be so restricted.

Professor MacCurdy says[3]: "Judging from the nature of the excreta of Permian fishes, their diet was exclusively carnivorous, but the evidence is faulty, as excrement of a vegetal nature might not be preserved." If the Permian fishes exhibited true caries and were wholly carnivorous, these facts would be a strong argument against the carbohydrate fermentation theory of Miller. As to the purely carnivorous diet, there is some doubt, however. In this connection, we note the herbivorous specialization of the teeth of some of the Paleozoic tetrapoda, successors of the Permian fish. Then too, it is possible that the caries reported was not true caries, but a phenomenon of a nature similar to that described by Howe in reference to his experiments with guinea-pigs on scorbutic diets—a softening of the teeth from within (apparently a leaching of lime salts back along the dentinal tubuli).

Concerning prehistoric man, MacCurdy in "Human Origins" (4) presents evidence of pyorrhea alveolaris and calculus in specimens of the Neanderthal race. The Rhodesian skull shows marked carious and pyorrheal destruction. Dr. C. E. Johnson (5) mentions that "a

[3] Personal communication.

considerable amount of alveolar bone is partially destroyed about the sockets of the teeth." This fossil was found at the Broken Hill mine, in Northern Rhodesia, Africa, in 1921. It is supposed by Sir Arthur Keith to be related to Neanderthal man, but by Dr. Woodward to be more recent. In reference to the dental conditions, Keith (6) says: "Until the discovery of the Rhodesian man we had always believed that caries or rotting of the teeth was a modern disorder, an ailment which civilized man had brought on himself by the artificiality of his diet.[4] It was therefore a surprise to find that this ancient man, with enormously strong jaws and big teeth, had suffered severely from dental caries and, we cannot doubt, from the pangs of toothache. . . .Of the sixteen teeth set on the upper jaw, ten are affected with this disorder. The remaining six, including in this group the missing incisor (and cuspid?) are worn but free from disease. At the roots of the molar and other teeth abscesses had formed and broken." He goes on to state that, on examining the teeth of wild chimpanzees which had lived and died in their native forests of tropical Africa, he found that they had suffered from caries, although only to a slight degree. This disorder, therefore, did not appear for the first time with the introduction of man to civilized life, although there can be no doubt that the discovery of cooking, and artificial ways of preparing food, have tended to increase this disease. The Australian aboriginee living on his native diet is almost free from caries, but he becomes subject to it when he adopts the white man's diet. Is it possible that the condition of the Rhodesian man's teeth represents the first fruits of the discovery of cooking? Or was it that he, like the Australian aboriginee, came into contact with an imported higher civilization that ultimately exterminated him?

In a study of 131 Peruvian (pre-Columbian?) skulls, containing 1259 teeth, Professor MacCurdy found 15.4 per cent to be carious and 13 per cent of all the jaws to be affected with pyorrhea. In the upper jaw of a youthful diminutive female about sixteen years old, the sockets of the molars showed evidence of severe pyorrheal involvement. Captain Leigh (7) found an incidence of 1 per cent of dental caries in

[4] Keith appears to have overlooked the fact that evidence of dental disease is found in the Neanderthal group.

395 Eskimo crania. He states that resistance to dental caries is greater in the Eskimo than in any tribe of American aboriginees. The early Sioux Indians, who also subsisted largely on an animal diet, were comparatively free from caries and from disease of the investing tissues of the teeth as well. They were a nomadic and very agile people.

The foregoing considerations lead to these conclusions:

(1) Dental disease, both caries and pyorrhea, occurred in primitive and even in prehistoric man, and the causes of dental disease are not, therefore, exclusively concomitant with civilization or the use of cooked foods.

(2) Dental disease has been enormously increased as a result of civilization.

Fifteen years ago the idea was prevalent that dental disease did not occur and could not be produced in the lower animals. This supposed fact was held to greatly increase the difficulty of research. Such opinion is not only untrue, but was contrary to the knowledge available at that time. Dental disease does occur in the horse, dog, and cat, (8, 9) and in rats and guinea pigs on stock diets (10). I have observed evidence of pyorrhea in the jaws of pigs, boars, and sheep. Dental disease can be produced in rats, guinea pigs, cats, dogs and monkeys, and doubtless in other animals. Some investigators prefer to describe their experimental lesions as "caries-like" and "pyorrhea-like." Our own observations lead us to regard the conditions induced in dogs as homologous with human dental disease. Therefore we may assert that dental disease occurs and can be experimentally induced in the lower animals.

Evolution as a factor in dental disease

Can susceptibility to pyorrhea (and dental caries) be inherited? This is a very important question. I could cite a number of cases where one-half the children of parents, one only of whom is afflicted with pyorrhea, present the same clinical type-picture as that parent. Doubtless most clinicians have made similar observations. I have in mind a gentleman of middle age, who presented a definite clinical picture of pyorrhea. His wife was free from this condition and slightly susceptible to caries. The son has large teeth, with enamel lobes well

coalesced, replicas of the father's teeth, but the gingivae showed unmistakably a hypersensitivity to irritation. Slight faults in hygiene evoked acute bloated conditions. The daughter was quite free from this tendency, resembling her mother. Racially, the prevalence of congested and swollen gingivae among Jewish people suggests that this susceptibility has gotten into the inheritance.

A case of indubitable inheritance of a dental character was observed in a patient affected with blue (water[5]) teeth. The color of these teeth, and the appearance of their tissue when studied by transillumination, was very distinctive, and evidently abnormal. Of the three children, one presents the same condition, another is normal, and the third, an infant, has not yet erupted any teeth. The mother can trace the condition back through her mother, grandfather, and great-grandfather. Thus, the character has run through at least five generations. The children's father and maternal grandmother were free from it. On the paternal side of the mother's descent, the occurrence of these blue teeth was divided equally among the offspring. There is little doubt that this case reveals a sport or mutation, which probably occurred quite suddenly in this family.

To revert now to a consideration of the prevalence of congested gums in Jewish people, there are several possible explanations, assuming, of course, that the observation is valid. *First*, the susceptibility developed as a result of slow evolution (the Darwinian type). To this answer there are two strong objections: it is very questionable whether even the antiquity of the Jewish line marks a sufficient lapse of time for selection (artificial) to have impressed this character upon the inheritance. Then, of course, we find ourselves in opposition to the law that acquired characters are not inherited. Besides, there are parallel data to show that dental disease may arise under conditions that would render mutations highly improbable in the course of a few generations. Thus McKay (11) has shown that a 50 per cent incidence of malocclusion has attacked the Sioux Indians in a few generations through contact with our civilization, and yet as far back as primitive skulls can be traced the arches were normal. He has also

[5] So designated by her family physician when she was a child. He had probably seen other such cases, since he had a name for the condition.

shown a serious prevalence of pyorrhea in the Pimas of Arizona—true pyorrhea occurring in children of fifteen or sixteen years of age. This susceptibility has been contemporaneous with their introduction to civilization.

As a *second* explanation, it is possible that susceptibility to gingivitis or pyorrhea among this race may have arisen as a mutation, but, *third*, the more probable explanation is to be found in the racial habits, especially the characteristic dietary practices of this people.

As concerns the inheritance of dental characters, (1) it seems not to be doubted that such a dental character as blue teeth is inheritable (in Mendelian ratio). (2) It seems probable that susceptibilities to pyorrhea are or get into the inheritance, (a) possibly through slow (artificial) selection (reversal of evolution through preservation of the unfit); (b) possibly through inherited variations in tissue chemistry—for example, such as lead to the necessity for blood groupings. Susceptibility of chemo-structural variants to acquired irritants might in this case be quite fortuitous.[6] Or to put it another way, there is no doubt that individuals showed different resistance to pyorrhea when first the species became afflicted with this disease. There is no good reason to suppose that these individual degrees of resistance might not be transmitted.

II. LOCAL EXOGENOUS PARODONTITIS

(*Schmutzpyorrhoe*)

What are the effects of abnormal environmental influences on the dental tissues? Among civilized mankind at the present time inflammation of the gums (gingivitis) is so common as to be almost universal. The clinical condition that most closely parallels the inflammation, both in position and (to some measure) in degree, is that of stagnation of debris about the necks of the teeth.[7] Over and over again, in the examination of all kinds and conditions of mouths, we find the in-

[6] Why not first, second, and third class structure of body as well as of other materials? Chemical evidence strongly suggests that individuals differ in quality of tissue as well as in form.

[7] After inflammation is established, contributions are made to this debris by seepage from the tissues themselves, thus establishing a vicious cycle.

vesting tissues good where the hygiene of the gingivae is good and involved where the hygiene is poor. Such conformity does not exist in the relation of other clinical irritants (e.g., traumatic occlusion, or faulty dental work, per se). Furthermore, removal of this debris and prevention of further stagnation in many cases relieves the inflammation.

As to the nature of this debris, it may be derived from the following sources: the secretions and excretions of the glands, possible exudates from the soft tissues, epithelial cells, microörganisms, possible regurgitative material, and remains of food. Of these various elements we are inclined to convict the last mentioned as the principal malefactor. However, does it not seem strange that an organ of the body should be so delicate as to become diseased through the lingering presence of substances to which it is accustomed at frequent intervals in the regular exercise of its function? If a concrete mixer, for example, were fabricated of such a material as would quickly disintegrate under particles of rock or cement that might lodge in crevices or corners, we would rightly regard it as unsuitable for its purpose. The answer would seem to be found in the abnormal nature of the food to which our teeth and gums are subjected as a result of the practice of cooking and of our civilized dietary habits. There is reason to believe that any ordinary lodgment of remains of uncooked and natural foodstuffs would not be so detrimental. In an effort to throw light on this problem, a sheep's head was procured and gum excised from a region where a considerable mass of food debris had lodged. Microscopic examination of this tissue revealed an absence of any sign of inflammation. On the other hand, an analogous section of a biopsy specimen from the mouth of a robust, healthy girl of 14 years showed marked reaction associated with the presence of *materies alba*.

What, then, is the relation of *materies alba* to superficial inflammation of the gingivae (and to caries)? Oran Starr, in a recent speculative work, attempts to relate dental disease to the laws of Lamarck and Darwin. Protoplasm, he says, does not tolerate either a sudden or radical change in environment. Such a change occurred in the transition from the use of uncooked to cooked foods. This is not the sole etiologic factor, for we have seen caries occur even where cooked

foods are not eaten, e.g., amongst wild animals. Opposed to Starr's theory also is the experiment of Howe (12), where, after feeding thirty guinea pigs for a year upon various sugars, starches, and microörganisms, no effect upon their teeth was discernible. Nothing is said by Howe as to the gums in these animals. We have also to consider that other conditions may not have favored stagnation about the teeth, even though a fermentable diet was used. In a recent examination of mature men who have been a year on a high carbohydrate diet, there was found convincing, although very slight evidence of increased caries. None was found to have developed during this period in men on a high protein diet.

It would seem, however, that the noxious effect of *materies alba* is dependent only in part upon remains of cooked foods, and is under the control of local complexes of ultimate systemic origin. Yet the fact that chemically and bacterially *materies alba* is irritating, and the close correlation of *materies alba* with inflammation, together with the fact that in many cases the persistent removal of debris relieves the inflammation, justify the diagnosis of many cases of superficial parodontitis as due to stagnation. But, as will be shown, inflammations of the gingivae may occur without the presence of debris; and so, while stagnation is one cause, it is not the only etiologic factor.

III. LOCAL ENDOGENOUS PARODONTITIS

We are now confronted with the question: Can inflammation of the gingivae occur in the absence of any external physical or chemical irritant (e.g., *materies alba*, trauma, or calculus)? That inflammation of the gingivae can be produced by purely endogenous agencies is demonstrated by the possibility of inducing experimental scurvy through a diet deficient in vitamine C.

Other cases, not scorbutic, but which conform to such types of periodontoclasia as the dentist would regard "true pyorrhea," may also be cited. A young woman, 22 years of age, applied some time ago for treatment of pyorrhea. The case was of particular interest because of the striking localization of the lesions. The teeth and investing tissues of the upper jaw were clinically perfect. In the lower jaw a pocket was found between the left first bicuspid and cuspid. Blood gushed from this region on the slightest touch. Deep

gullies were observed in the alveolar process (radiographic data) distal to the right first bicuspid and right first molar. The parodontia of the anterior teeth were ravaged—only the very ends of some of the roots remained embedded in bone. The remainder of the tissues of the lower jaw were good. There was no history of Vincent's infection. The teeth were carefully scaled and perfect coöperation obtained from the patient in brushing the teeth and gums. She was put on a presumably optimal diet, containing an abundance of vitamines and mineral salts, and the acid-base balance was adjusted to conform with our most recent ideas. Cod liver oil was also prescribed. Aspiration from the gums, according to the method of Beckwith (13), yielded a pure culture of *Staphylococcus albus*. An autogenous vaccine was prepared from this and used in the customary dosages.[8] The patient, well educated and intelligent, is vitally interested in her teeth. She appears to be an accurate and reliable observer. According to her account, the trouble in her lower front teeth was ushered in by a burning sensation, followed by a reddening and puffiness of the gum margin. Following this, she noted in a gingival papilla what she described as a bright, red line. When these symptoms subsided, the gum seemed to slump a little.

Several months after the case was started and when treatment was at its height (scaling, hygiene by patient, massage, optimal diet, cod liver oil, and vaccine) she complained of a sensation similar to that described, distal to the upper right cuspid, indicating with her finger the position, which she could not readily see and had probably not seen. On examination I found a dilated capillary in the gingival papilla—a bright red line looking like a silk fibre in a greenback. Now, according to the data of Fleming, this is one of the least susceptible regions in the mouth. There was no detachment. Contact, embrasure, and occlusion were normal. There was no trauma, no recent injury, and the hygiene, as far as could be judged clinically, was perfect—certainly as clean as could be found anywhere in a human

[8] I have recovered organisms in certain cases, yet on repeating the aspiration have gotten no growth, even where the operations were carried on in fields that were congested or bloated and where blood was washed back into the syringe. I did not repeat this case, and am not at all sure that the finding of this organism is particularly significant. On a second trial I might have gotten nothing.

mouth. This occurred a few weeks before the time of the International Dental Congress at Philadelphia (August, 1926). When the patient returned a month later, it was found that another area (the gingival papilla between the upper central incisors) had developed precisely the same symptoms—burning, puffiness, and an injected capillary. We continued our treatment and no further eruptions have occurred, whether due to spontaneous arrest or to some factor in the treatment I do not know. Further data on this case, compiled by Dr. H. C. Hinshaw, are of great interest. In protozoological studies he found an entire absence of amoebae and trichomonas. The exudate was largely made up of polymorphonuclear leucocytes, with *very few bacteria*—almost a sterile pus.

The possibility of other endogenous gingivitides has been shown by the results of aspiration and culture from certain cases. In one case, where the gums were so bloated that introduction of a hypodermic needle caused the blood to squirt out, we secured no growth on culturing fluid aspirated from this tissue. In another patient, whose gums presented great areas of liver-like consistency and the color of currant jelly,[9] aspirated material again produce no growth. Consequently, it would appear (1) that superificial gingivitis may be produced by purely endogenous causes, and (2) that non-infectious inflammations may occur, e.g., possibly analogous to angioneurotic edemas. Alternative conjectures are, infection by filtrable viruses or non-invading surface pathogens.

IV. PARACEMENTITIS: INFLAMMATORY PERIODONTOCLASIA

Following gingivitis may occur either horizontal or vertical involvement of the deeper tissues. It would seem that the horizontal type is merely the sequel of chronic gingivitis uncomplicated by susceptibility. Horizontal paracementitis of purely local origin is well illustrated by sialocalcic periodontoclasia. It is the sequel of sialocalcic gingivitis. A type case has been described in detail in a paper entitled, "The etiology of parodontoclasia (14). In this case the etiologic factor was accretion of salivary calculus. The result was a slow

[9] An apt analogy employed by Dr. W. H. Hanford.

wasting away of the parodontia at right angles to the long axes of the teeth.

Intermediate between the horizontal and vertical forms is the clinical type—sulcicalcic parodontitis. This condition is characterized by relatively shallow pockets, generally less than four millimeters, and deposits of calculus within the pockets. The inflammation is intense, the gingivae and alveolar mucosa swollen and engorged, and resistance active. The etiologic factors are inflammation, combined with dehiscence, which is a splitting away of the soft tissues from the teeth, related to downgrowth of epithelium. The nature and possible cause of this phenomenon will be discussed subsequently.

In cases where the tissues are unable to oppose sufficient resistance to exogenous irritants, or where local endogenous or systemic susceptibilities exist, vertical extensions may occur. Clinically we can distinguish two types—the crevicular and granulative. The former is characterized by pockets often deep and presenting narrow longitudinal extensions. The latter is characterized by deep pockets, and marked widening of the pericementa through resorptions of the alveolar walls. It appears to be a complex condition due to the superimposing of inflammation upon a lateral halisteretic parodontal osteoclasia (14).

Microscopic study reveals two possible pathways for the spread of inflammation from the gingivae. One is along the pericementa, the other through the bone marrow.

Both the tissue of the marrow and the fiber of the pericementum act as barriers to the inflammatory process. The relative strength of these barriers, anatomic and histologic details of structure, and the location of the portal of entry of infection, determine the relative involvement of the bone marrow on the one hand and of the pericementum on the other. In some cases the bone marrow is ravaged, while the pericementa remain quite good. In other cases the reverse is true. The specific factors that determine the variable resistivity of these tissues are at present unknown, although there is some evidence that rapid invasion of bone marrow is associated with a lowering of biologic defenses through certain systemic illnesses.

V. PARACEMENTOSIS: NON-INFLAMMATORY PERIODONTOCLASIA

Of all the types of periodontoclasia, that characterized by a resorption of the alveolar process, without the necessary presence of any inflammation, has been most obscure. In this country investigators as well as clinicians seem to have overlooked it, in spite of the fact that it constitutes a definite and easily recognizable entity, is not at all rare, and seems to merit attention as a result of the writings of Talbot and Hopewell-Smith. Gottlieb has described the histologic picture of this condition under the head of "diffuse atrophy." He has reached the conclusion that it is of systemic origin, but not related to any systemic disease. Evidence that this condition may be produced experimentally in the dog, due to a disturbed acid-base balance of the diet, has been advanced in an earlier publication (15), from which the following is quoted:

"(1). The non-inflammatory type of parodontoclasia has been observed in dogs placed on diets in which basic ions were in excess. A reversal of the dietary fault has brought about osseous reconstruction.

"(2). The microscopic appearance of the retrograde tissues resembles diffuse atrophy such as described by Gottlieb in human tissue.

"(3). The retrograde conditions occurred in cases where there were no degenerative changes in the long bones, and where the animal remained apparently in good health, robust, and well-nourished. This is in conformity with the occurrence of parodontoclasia in humans who seem to be in perfect health.

"(4). The retrograde bone changes observed, namely, resorption of the crest of the alveolar septum, lateral excavations, opening of the bone marrow spaces to the side, osteoporosis, coalescing of marrow spaces and widening of the sockets of the teeth crownwise, all compare with the appearances familiar to parodontists in roentgenographs of human parodontoclasia."

The parodontal osteoclasia may appear as halisteresis, and may be accompanied by osteoporosis. Halisteretic changes show a predilection for subperiosteal bone, especially that most recently formed. They involve the crests of the septa first, but may appear also along the lateral margins. In the early stages of osteoporosis, the decalcification seems to be effected by mononuclear osteoclasts, which gather in files two and three layers deep. The later resorptions, as well as the

removal of the matrix left by halisteresis, are associated with the appearance of multinuclear osteoclasts (giant cells). The two types of osteoclasts show a marked tendency to occupy different loci at any given period, and the same locus at different periods. Arey (16) believes that the chief source of osteoclasts is from old osteoblasts and bone cells—that depleted, basophilic osteoblasts coalesce to form multinucleate masses, and these syncytial elements become typical osteoclasts, their cytoplasm assuming an oxyphilic stainability.

Lacunar resorptions and Volkmann canals may occur both in physiologic and pathologic conditions. It is sometimes very difficult to ascertain which is the case. Bone is a labile tissue, and especially is this true of the alveolar process of the jaws. Destructive and constructive changes are constantly in process. If, however, the regressive changes are in excess of those to be accounted for on the basis of architectural and stress rearrangements, and become dominant, the condition will be pathologic. From a clinical standpoint, we are largely dependent upon the roentgenograph for diagnosis of the bone changes in parodontosis. It is hoped that in the future comparative roentgenographic and microscopic studies may render possible more refined differential diagnosis. At present, however, it is desirable to exercise caution in basing dogmatic assertions on roentgenographic evidence, for we know that similar alveolar patterns may result from quite different causes.

VI. THE DEHISCENCE OF THE CUTICULA DENTIS, AND THE DOWNGROWTH OF EPITHELIUM

The studies of Gottlieb have shown that the gingival sulcus (pocket, as he calls it) is not the entity that has been previously understood. As the tooth erupts it carries with it the remains of the enamel organ (an epithelial structure). When this reaches the epithelium it coalesces with it. As the tip of the tooth appears at the surface, an opening is formed. At this time the epithelium of the mucosa is continuous with the remains of the enamel organ (Nasmyth's membrane), and the latter is securely affixed to the enamel. There is no sulcus. As the tooth moves upward into occlusion, a dehiscence of the gingival epithelium from the enamel cuticle takes place, and this dehiscence forms the true sulcus. It has been previously thought that when the

crown of the tooth had come into occlusion, eruption was complete, and the bottom of the sulcus was at the gingival line. It would appear that the exact significance of epithelial downgrowth is *sui generis* at the present time.

We have recently obtained, through studies of monkey, coyote, and opossum tissue confirmatory evidence of the physiologic nature of the cemental *Epithelansatz* in the region of the neck of the tooth. There is some evidence that disease may predispose to acceleration of this process.

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THE AMOEBAS OF THE MOUTH IN RELATION TO INCIDENCE OF PYORRHEA, CLINICAL SYMPTOMS, EXTENT OF LESIONS, AND AGE OF PATIENTS[1]

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In order to obtain an intelligent and comprehensive understanding of a disease it is necessary to study thoroughly the rôles of various factors. The present report deals with one of the problems of oral protozoology—the relation of amoebic infection to clinical symptoms.

I. MATERIAL AND METHODS

Material upon which the present research was done was obtained largely from three sources. San Quentin Prison offered an unlimited

[1] Presented at a session of the Sixth Annual Meeting of the International Association for Dental Research, Hotel Mayflower, Washington, D. C., March 25–28, 1928.

quantity of material. One hundred and ninety-one inmates of this institution have been utilized. However, the number of variable factors and the magnitude of variations made this group undesirable for precise work, especially in regard to mouth hygiene, age, history, etc. Many of the men questioned had never used a tooth brush. Very few made use regularly of this toilet necessity. Many nationalities and several races were encountered. For these reasons a different group was utilized for more detailed studies. We thought that university students would constitute a more uniform group. With the coöperation of Dr. T. W. Cook, of the Dental Infirmary of the University of California, we procured material from 57 students at the time of the dental examination made upon matriculation. But the hygiene factor was far from uniform in this group, the students were inaccessible for successive studies, and sufficient time could not be taken for an adequate periodontal examination of each. Students in the College of Dentistry of the University of California, in San Francisco, constituted a group well informed in dental matters, whose hygiene was exceptionally good on the average, who were at all times available for re-examination, and who were thoroughly coöperative in every respect. In this paper the one hundred cases reported upon were from this group. Material has also been obtained from ten other individuals, most of whom were faculty members in the University.

Prisoners in San Quentin were examined superficially for the most part and brief notes taken. The wide variety of lesions present in their mouths, and the probability of many factors operating in the production of these lesions, indicated the necessity for devising a classification suitable to our problem. Advanced cases included those presenting typical profound pyorrhea, with all of the cardinal symptoms. The gums were usually retracted and the interdental papillae destroyed. There was macroscopic pus with other signs of deep seated, chronic inflammation. Often the teeth were palpably loose as the result of the destruction of the alveolar process. Subgingival calculus was present, often in large amounts. Most such mouths were excessively foul and poorly cared for. These were cases of undoubted generalized pyorrhea. At the opposite extreme could be readily recognized those mouths which were on the whole clean, and

free from advanced pyorrhea as usually diagnosed. Not infrequently it would be noticed that gums were prone to bleed. Relatively shallow gingival pockets might be present, together with small deposits of subgingival calculus and microscopic pus. Thus the incipient stages of pyorrhea might exist, but the involvement was so slight that many dentists doubtless would have considered all of these as within the normal. We recorded them as such on our records. Intermediate between these were a number of individuals who could not safely be classified either as relatively normal or as possessing typical pyorrhea. In several, inflammation was directly traceable to large accumulations of putrefying debris. Others appeared to have a form of pyorrhea at least temporarily quiescent or inactive. Some lesions could be traced to the ravages of syphilis, Vincent's stomatitis, tuberculosis, or other diseases. Some were borderline cases, which could not be accurately classified in the time available and under the conditions. We shall refer to this entire group as constituting the uncertain and anomalous cases.

The students in dentistry at the University were examined with considerable care. A chart was used with spaces for recording the following data more or less quantitatively: gingival recession, pericemental detachment, pocket formation, the presence of pus, loose teeth, gingival hypertrophy, subgingival calculus, supragingival calculus, detritus, caries, fillings, extracted teeth, and inflammation. There were four columns, in each of which could be placed + or ++. These columns were labelled: "none," "slight," "moderate," "marked." There was also space for indicating whether the gums were of a "marked red color," "marked purple," "slightly inflamed," "inflamed gingival margin only," "anaemic," "normal." In addition to the patient's name, we noted his age and nationality, together with data concerning recent periodontal treatment and a description of the gross physical state of the saliva. Rather copious notes were usually made describing the general appearance of the gums, and a description of the appearance of the sites from which cultures and smears were made. If anything worthy of note was learned about the general health of the individual, or his dietary preferences and practices, these were recorded. We also inquired as to the frequency of brushing the teeth.

1. Preparation of stained slides

Smears were made in all cases. Subgingival exudate, or when this could not be procured, interproximal debris was obtained with a dental scaler and quickly transferred to a clean thoroughly flamed slide on which had been placed a very small drop of blood serum preserved with thymol. The material was thoroughly teased out in the serum, then quickly smeared into a thin layer, and, before drying could take place, rapidly plunged face downward into the fixing fluid. The use of blood serum as a fixative should be stressed here. The coagulum formed upon contact with fixing fluid is much more adherent to the glass slide than that of egg albumen, and the strands of coagulated protein are sufficiently fine to be entirely unobjectionable. The coarse strands of egg albumen take the stain and frequently obscure valuable material.

The fixing fluids employed were Schaudinn's alcoholic sublimate, Bouin's picric acid-formalin mixture, and Zenker's bichromate-sublimate mixture. The first two were usually employed in routine work. Staining of smears was generally accomplished by means of Heidenhain's iron haematoxylin method. None of the dry smear methods so commonly used are dependable for diagnosis.

2. Diagnosis by the culture method

It was found that the culture method was an essential procedure if we were to detect cases of light infection with *Endamoeba gingivalis*. Our method was essentially that used by Howitt (1925), employing a slight modification of the medium devised by Boeck and Drbohlav (1925). This consists of a coagulated slant of whole egg slightly diluted with Locke's solution covered with a larger amount of dilute egg albumen in Locke's solution. The use of small 1 cm. x 10 cm. tubes for field cultures was found to be very convenient and thoroughly as effective as, if not superior to, large tubes. Pockets can be made inside a man's vest to hold as many as 50 of these tubes. In this location a temperature approximating that of the body may be maintained, and the tubes present no inconvenience whatever. Tubes were often carried 8–10 hours in such pockets with perfect safety and excellent growth of the amoebas.

After 48 hours' incubation, two or three drops of the sediment collected in the bottom of the tube was examined under the microscope. During this time the few amoebas inoculated had multiplied sufficiently to make their detection a simple matter. If examined in a warm room, using warm slides, the motility of the amoebas is maintained for at least 10 to 15 minutes. With a warm stage motility will last even longer. The animals are readily recognized by their waxy, greenish, refractile, protoplasm and their locomotion by means of broadly rounded, clear, pseudopodia. Detection is much simpler than in fresh smears made directly from the mouth. Not only are the amoebas increased in number, but the leucocytes have disintegrated and other confusing debris has largely disappeared. The culture method is not infallible, however, and negative cases were recultured and additional stained smears were made. In cases of failure to obtain a satisfactory culture, when stained smears proved that the organisms were present, it was usually possible to trace the disturbance to the presence of some antagonistic organisms in the culture. In cultures made of material taken from very foul mouths, there may develop proteolytic putrefactive bacteria in excess, making it impossible for the amoebas to exist. The egg slant is sometimes blackened by the activity of these organisms, and such cultures are usually negative. *Trichomonas buccalis*, a flagellate parasite common in advanced pyorrhea, is definitely antagonistic to *Endamoeba gingivalis* in vitro. Amoebas are seldom numerous in the presence of this flagellate, and may be absent although stained smears of the exudate reveal numerous amoebas. It must be emphasized that both methods, smear and culture, are essential to any thorough survey if all, or perhaps even if most, of the positives are to be detected.

II. OCCURRENCE

The literature dealing with the protozoa of the human mouth is badly confused with widely differing reports as to the conditions in which these parasites occur. Divergent opinions exist as to the scope of the term "pyorrhea." In consequence we dare not accept the statement that protozoa occur in the "normal" mouths unless we know what the author's conception of "normal" is. Too frequently he means the "average" mouth. To overcome this difficulty we have

entered into a rather detailed study of the exact conditions in which these organisms are found, expressing the degree of involvement in quantitative terms when possible.

1. Relation to depth of gingival pocket

There can be no doubt that, in the diagnosis of the extent of inflammatory destruction of the tissues investing the teeth, the measurement of dental detachment is an invaluable method. In the absence of a radiographic record it is the best method of diagnosis of pyorrhea. Since there is evidence that atrophic changes in the alveolar process may take place from purely endogenous processes, we regard this method as superior to roentgenographic evidence in evaluating the rôle of parasitic microorganisms about the teeth.

The method has been described by Simonton (1925), who made the examinations upon which we are reporting. Time did not permit the careful exploration of the gingival sulcus on all sides of each tooth, but a sufficient number of regions were studied to get a good idea of the extent of destruction. The dental students who were examined usually had a pretty good idea of conditions in their own mouths, and were often able to direct us to the most involved area at once. Having secured an idea of the amount of peridental membrane destroyed, we made upon our record one of the following entries: no detachment, slight +, slight ++, moderate. In the group characterized as possessing "no detachment," we included only those with a maximum detachment of less than 0.5 mm. It was scarcely possible to estimate more closely than this. Those with a detachment referred to as "slight +" possessed at no point a loss of more than 1 mm. of peridental membrane. Those recorded as possessing a detachment of "slight ++" had lost from 1 to 2 mm. of peridental membrane in the regions of greatest involvement. Those characterized as having suffered "moderate" detachment had lost more than 2 mm. of peridental membrane in those regions where destruction had proceeded most extensively. When the gums had receded below their normal height, this measurement was subtracted from that of the detachment to give the depth of the gingival pockets.

Table 1 summarizes our results, which have been published (Hinshaw, 1926) and are graphically illustrated in *fig. 1*. *Nineteen of the*

20 cases of amoebic infection, or 95 per cent, had pockets over 0.5 mm. deep, while only 37 of the 80 free from infection, or 46.25 per cent, had pockets over 0.5 mm. and none extended beyond the depth of 2.0 mm. *It will be observed that among those relatively free from gingival pockets, very few if any harbor this parasite.* The single case giving a positive culture among the 44 examined in this group was not normal in all respects. There was very slight detachment (less than 0.5 mm.) in the region of the upper first molars. The gum tended to bleed upon exploration and had receded slightly. A single amoeba was observed in the first culture. None were seen in the smear, although it was carefully studied. Two successive examinations were made, at intervals of two weeks and seven weeks respectively, and no amoebas could be recovered either in smear or culture. We consequently

TABLE 1

Relation of amoebic infection to depth of gingival pockets

MAXIMUM DEPTH OF GINGIVAL POCKETS	0–0.5 MM.	0.5–1.0 MM.	1.0–2.0 MM.	OVER 2.0 MM.
Number infected with *E. gingivalis*	1	6	4	9
Percentage of infection	2.27	15.0	57.14	100
Number free from protozoan infection	43	34	3	0
Percentage not infected	97.73	85.0	42.86	0

concluded that this was an extremely light infection or possibly only a temporary one.

While the clinical data are not so complete in the cases examined at San Quentin Prison and elsewhere, we can state that we have never found an infection with *Endamoeba gingivalis* in the absence of gingival pockets. In general, results were obtained analogous to those secured from students in dentistry. There is one important fact to be noted in this connection. It is not likely that our results could be exactly duplicated upon the general population or upon any less uniform group. It is quite possible that an agent operating for fifty years would produce a pocket of the same dimensions as that made by a more rapid agent working through perhaps ten years, and the flora and fauna of the two pockets would differ while their measurements would not. We have met with a few older individuals, with pockets between 2.0

and 3.0 mm. deep, who were not infected with amoebas. We have never found amoebas in a normal mouth, old or young, among the 357 persons examined.

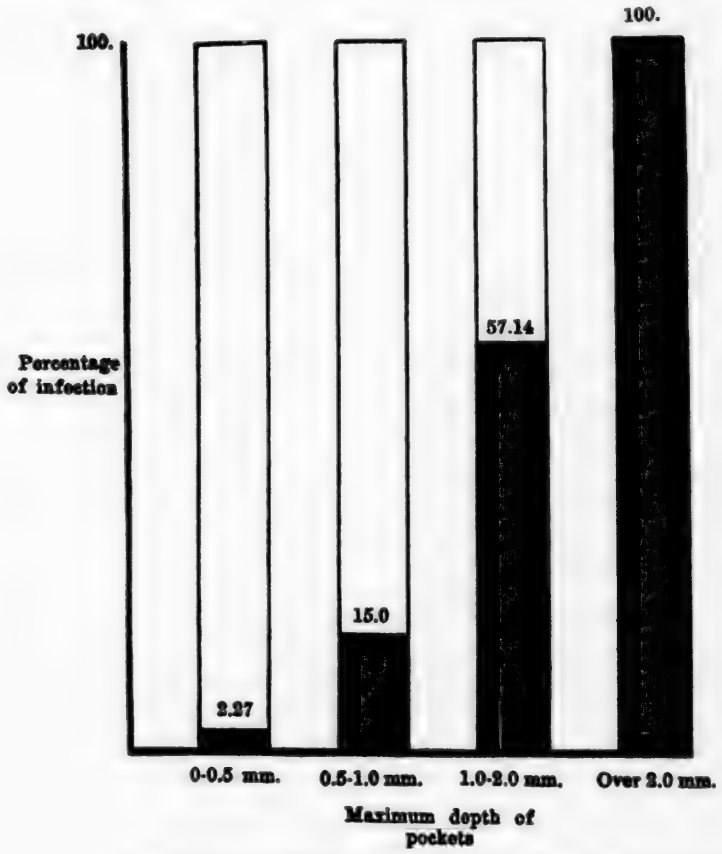

FIG. 1. Relation of amoebic infection to depth of gingival pockets. The height of each black column represents the incidence of infection in the group. The extent of gingival pocket formation is indicated at the base of each column.

2. *Relation to recession of the gum*

In our records we expressed recession as "none," "slight +," "slight ++," "moderate." Numerical equivalents would closely parallel those chosen for estimation of the depth of gingival pockets. The recession of the gum is frequently associated with pocket formation, and like the latter is a measure of the extent of retrograde processes. Table 2 summarizes the results of this comparison among 100 dental students. It will be seen that 19 of the 20 cases of amoebic infection, or 95 percent, manifested at least some recession, while 66 of the 80 persons free from infection, or 82.5 percent, showed recession.

TABLE 2

Relation of recession of the gums to infection with E. gingivalis

AMOUNT OF RECESSION	NONE	SLIGHT +	SLIGHT ++	MODERATE
Number infected with *E. gingivalis*	1	12	6	1
Percentage of infection	7.14	16.9	42.86	7.14
Number free from protozoan infection	13	59	8	0
Percentage not infected	92.86	83.1	57.14	

TABLE 3

Relation of hypertrophy of the gum to amoebic infection

AMOUNT OF HYPERTROPHY	NONE	SLIGHT
Number infected with *E. gingivalis*	13	7
Percentage of infection	16.7	31.8
Number free from protozoan infection	65	15
Percentage not infected	83.3	68.2

We examined several older individuals with recession amounting to several millimeters, but with very shallow pockets or no pockets at all, who were negative for protozoa. There are many factors which may produce recession of the gum without working sufficient damage to cause pyorrhea. One of these and of major importance is the use of a stiff toothbrush, especially if the stroke is laterally directed with considerable pressure and if long continued. This type of recession is especially to be noted on upper lateral incisors and cuspids. It bears no relation to pyorrhea, and does not furnish favorable ground for protozoan infection.

3. Relation to hypertrophy of the gum

Hypertrophy of the gum is sometimes met with in a slight degree where pyorrhea does not exist. We were interested to determine if amoebas were living in such areas, and particularly if hypertrophy were usually associated with amoebic infection. The results, tabulated below (Table 3), *indicate that hypertrophy is relatively rare in early stages of pyorrhea and amoebas are not more frequent in slightly hypertrophied gums than in gums of normal size.* In fact, 13 of the 20 cases of amoebic infection, or 65 percent, showed gums of normal size, and 65 of the 80 not infected, or 81.25 percent, were of normal size. In San Quentin Prison we have occasionally seen greatly hypertrophied gums in ill kept mouths with no amoebic infection. *We therefore conclude that amoebic infection is not associated with hypertrophic growth of the gingivae.*

4. Relation to type of inflammatory response

While we cannot state with certainty that *Endamoeba gingivalis* calls forth any inflammatory response, we can determine something of the condition in which it lives, and can state definitely that certain common inflammatory reactions are *not* caused by this organism. Even a casual examination of any considerable number of mouths will impress the observer that there are a number of types, and many degrees of inflammation, to be seen, some of which are directly traceable to poor dentistry. Poorly fitted crowns, overhanging fillings, and various appliances may lead to inflammation. We have secured cultures and smears from several such areas, but have never found amoebas when their presence would not be suspected from conditions elsewhere in the mouth. *If the remainder of the mouth is normal, amoebas are invariably absent from areas of traumatic injury.*

One of the common and most distinctive types of inflammation is that wherein a localized area near the gingival margin exhibits slight edema and a smooth glistening surface, with an obliteration of the stippled, normal appearance. There is usually an hyperemia in this region of edema, often a bright red in color. The area is generally in the form of a crescent, which may extend to the extreme gingival margin; but occasionally the inflammatory area lies 1 or 2 mm. from

the gingival margin, leaving a sharply defined marginal strip of less inflamed gum near the neck of the tooth. The neck of the tooth in contact with this type of gum is often covered with *materia alba.* Although this is a common condition, we rarely get cultures of amoebas from such localities; and then only when there are gingival pockets and suppurating gums elsewhere. On a few occasions we have taken cultures from such an area, and from other areas in a pyorrheal mouth, finding amoebas in all parts of the mouth except the region of the crescents of acute inflammation. *We therefore conclude that this common type of inflammation is not caused by Endamoeba gingivalis, but by another organism that is probably antagonistic to the growth of the amoeba. We have never found amoebas in mouths free from all inflammation, and very seldom in non-inflammatory areas of mouths with localized pyorrhea. It is probable that they multiply only in an area of suppuration.*

Pyorrheal pus consists in large part of polymorphonuclear leucocytes, some of normal appearance and some disintegrated, only the nuclei remaining. The latter are frequently spoken of as the "salivary corpuscles." If the number of normal leucocytes be counted in a number of microscopic fields of a stained smear, and also the number of denuded nuclei in the same area, and comparison made between two cases, one infected with amoebas and the other not infected, interesting figures will develop. *In amoebic infection there will usually be observed many more living leucocytes than disintegrating ones, while the reverse is likely to be true in the case not infected.* The number of disintegrating leucocytes is to a certain extent a reciprocal of the rate of pus flow. Obviously where leucocytes are being rapidly extruded, they will be eliminated from the gingival sulcus before large numbers die and disintegrate. Since the relative number of living leucocytes is probably a measure of the rate of pus flow, the proportion described seems to characterize amoebic infection. *It should be stressed that while pus cells may be present when amoebas are absent, they are always present when amoebas are present, and usually they are present in much greater numbers.*

The macroscopic appearances in amoebic infection vary considerably, but there is always a generalized hyperemia, usually in the form of a purplish venous stasis. The gums are often quite tender to explora-

tion, and always tend to bleed freely upon slight injury such as is produced in getting subgingival material for cultures, or even in brushing the teeth briskly.

5. Relation to supragingival calculus

It was not easy to ascertain the rate of deposit of this salivary or supragingival calculus upon the teeth of the dental students examined, because of the fact that most of the students have such material removed at rather frequent intervals. To find calculus present often

TABLE 4

Relation of supragingival calculus to infection with E. gingivalis

AMOUNT OF SUPRAGINGIVAL CALCULUS PRESENT	NONE	SLIGHT +	SLIGHT ++	MODERATE
Number infected with *E. gingivalis*	1	16	2	1
Percentage of infection	8.33	20.25	40.0	25.0
Number free from protozoan infection	11	63	3	3
Percentage not infected	91.67	79.75	60.0	75.0

TABLE 5

Relation of amoebic infection to subgingival calculus

AMOUNT OF CALCULUS PRESENT	NONE	SLIGHT
Number infected with *E. gingivalis*	18	2
Percentage of infection	19.15	33.33
Number free from protozoan infection	76	4
Percentage not infected	80.85	66.67

meant merely that it had not been removed as recently as in the case of another individual possessing no such deposit. From Table 4, giving our findings on this subject, it will be observed that 19 of the 20 cases of amoebic infection, or 95 percent, possessed at least some supragingival calculus. The remaining individual had had his teeth scaled less than one month previously. Of the 80 cases not infected with amoebas, 11 or 13.75 percent had no supragingival calculus. It thus appears that supragingival calculus may frequently be present in the absence of amoebic infection, but that amoebas tend to be found in a mouth with supragingival calculus.

6. Relation to subgingival calculus

The existence of subgingival calculus can only be determined by means of an exploring instrument, and failure to find small existing deposits may occur through oversight. For that reason we cannot assume that every individual whom we reported to be free from subgingival calculus really lacked small deposits of this material. Relatively few of the dental students are reported with such deposits, owing in part to the frequence with which the material is removed by instrumentation. Our report is contained in Table 5. The number

TABLE 6

Relation of amoebic infection to amount of detritus present

	NONE	SLIGHT +	SLIGHT ++	MODERATE
Number infected with *E. gingivalis*	0	16	2	2
Percentage of infection	0	25.0	13.33	15.38
Number free from protozoan infection	8	48	13	11
Percentage not infected	100	75.0	86.67	84.62

TABLE 7

Relation of amoebic infection to frequency of brushing the teeth

FREQUENCY OF BRUSHING	ONCE PER DAY	TWICE PER DAY	THREE TIMES PER DAY	NOT REGULARLY	NOT STATED
Number infected with *E. gingivalis*	3	8	1	1	7
Percentage of infection	13.04	16.0			
Number free from protozoan infection	20	42	2	0	16
Percentage not infected	86.96	84.0			

of positive reports is hardly large enough to be of statistical significance. *We are certain that there is a very important relation between subgingival calculus and E. gingivalis in later stages of pyorrhea, as is shown by histological evidence presented by Kofoid and Hinshaw (1928).*[1] This deposit, together with amoebas, invariably occurs in later stages of pyorrhea.

[1] Kofoid and Hinshaw: Proceedings of the International Association for Dental Research; *Journal of Dental Research*, 1928, viii, p. 446.

7. *Relation to amount of detritus present*

There is a widespread opinion that *E. gingivalis* is strictly a saprozoic organism feeding upon detritus in the mouth. Our results do not confirm this opinion (Table 6). *Amoebas were never observed in the absence of detritus. When amoebas were present, this material was generally of the nature of a cellular exudate, usually relatively scanty in amount.* It is significant that 16 of the 20 cases of infection, or 80 percent, exhibited only very slight amounts of debris. If this organism were strictly saprozoic, one would expect to find infection more

TABLE 8

Relation of amoebic infection to caries

AMOUNT OF CARIES	NONE	SLIGHT +	SLIGHT ++	MODERATE
Number infected with *E. gingivalis*	11	7	1	1
Percentage of infection	22.92	16.28	20.0	25.0
Number free from protozoan infection	37	36	4	3
Percentage not infected	77.08	83.72	80.0	75.0

TABLE 9

Relation of amoebic infection to past history of caries

NUMBER OF FILLINGS	NONE	SLIGHT +	SLIGHT ++	MODERATE	MODERATE ++	MARKED
Number infected with *E. gingivalis*	1	5	5	8	1	0
Percentage of infection		22.73	17.86	20.51	14.29	
Number free from protozoan infection	0	17	23	31	6	3
Percentage not infected		77.27	82.14	79.49	85.71	

frequent in mouths containing considerable amounts of debris. In those free from infection 56 of the 80, or 70 percent, had little or no debris. It is evident from these data that the distribution of amoebas in fairly clean and in dirty mouths is a random distribution, with the exception that they are not found in perfectly clean mouths.

Our work at San Quentin Prison, on a large number of men, has confirmed this conclusion. When there are large accumulations of food debris, and gingival pockets are absent, there may be a great deal of inflammation, often with a bright red arterial hyperemia, but

in these cases amoebas are not encountered. *We conclude that debris alone cannot support this organism.*

8. Relation to hygiene

Each person was questioned as to the number of times his teeth were brushed each day. It should also be mentioned that the dental students knew how to brush their teeth effectively. Our data are not complete on this point, but show *that there is little relation between infection with this parasite and habits of oral cleanliness* (Table 7). It will be observed that 9 of the 14 persons, or 64.29 percent of those infected with the amoebas on whom we have these data, brushed their teeth twice a day or more frequently. Of the 64 individuals not infected, 44 or 68.75 percent, brushed their teeth at least twice each day. We conclude that *E. gingivalis* may be found frequently, even when intelligent and persistent efforts at dental hygiene are expended.

9. Relation to caries

The data in Table 8 indicate that any chemical alterations which the saliva might hypothetically undergo in caries does not affect the amoebas to any considerable extent.

From the observation of one or two cases it appeared possible that an epidemic of caries within the mouth might be followed after a time with incipient pyorrhea. As an index to the extent of caries in past time, the number of fillings was taken. Under the heading, "slight +," was included all those with one or two very small fillings; "slight ++" indicates one very large filling or two medium sized ones; "moderate +" indicates about three fair sized fillings; "moderate ++" indicates about four such fillings; "marked" indicates about five or more. The data obtained (Table 9) give an almost ideal random assortment distribution, with *no evidence of a correlation between a history of dental caries and infection with Endamoeba gingivalis.*

10. Relation to gross physical states of the saliva

In each case we noted whether the saliva was plentiful or scanty, and watery, frothy, stringy, or glairy. These data were usually determined by pulling the lower lip forward from the teeth and noting the consistency of the saliva and its tendency to form sticky strings.

See Table 10. In cases of amoebic infection there is usually either plenty of saliva, or the saliva is of the adhesive type that clings to mucous surfaces forming a glairy film. The mucous surfaces of such mouths are continually moist, offering a favorable substratum for the amoeba. It is noteworthy that frothy saliva does not seem to permit the growth of *Endamoeba gingivalis*. The single case of infection in thin, stringy saliva was in the person who admitted that he did not brush his teeth regularly. In the group whose saliva was characterized as thin and glairy we also noted a slight tendency to become stringy in a very few instances. It is probable that these variations

TABLE 10

Relation of amoebic infection to gross physical states of the saliva

STATE OF SALIVA	THIN AND SCANTY	THIN AND PLENTIFUL	FROTHY	THIN AND STRINGY	THIN AND GLAIRY
Number infected with *E. gingivalis*	6	6	0	1	6
Percentage of infection	10.53	100	0	8.33	33.33
Number free from protozoan infection	51	0	7	11	12
Percentage not infected	89.47	0	100	91.67	66.67

in the gross physical appearances of the saliva are indicative of rather deep-seated chemical alterations, which may profoundly affect the organisms growing in the mouth. There should be an interesting field in the study of the biochemical qualities of saliva as related to the fauna and flora of the mouth.

11. Relation to local and systemic medication

We have noted that local medicaments, such as iodine, do not eradicate *E. gingivalis*. Habitual users of chewing tobacco and inveterate smokers are as likely to harbor amoebas as those not using tobacco. Men who were habitual users of such narcotics as opium and allied drugs have been cultured upon entering San Quentin, and found to be heavily infected with *E. gingivalis*. A large number of luetic prisoners have been studied both before and after receiving the chemotherapeutic treatment for syphilis. The treatment they received consisted of the intramuscular injection of 0.9 g.

of neosalvarsan at intervals of about one week. Combined with this were mercury rubs consisting of the application of about 0.5 g. of blue ointment applied to the axilla, elbow, and behind the knee every day for six days, omitting this treatment each alternate group of six

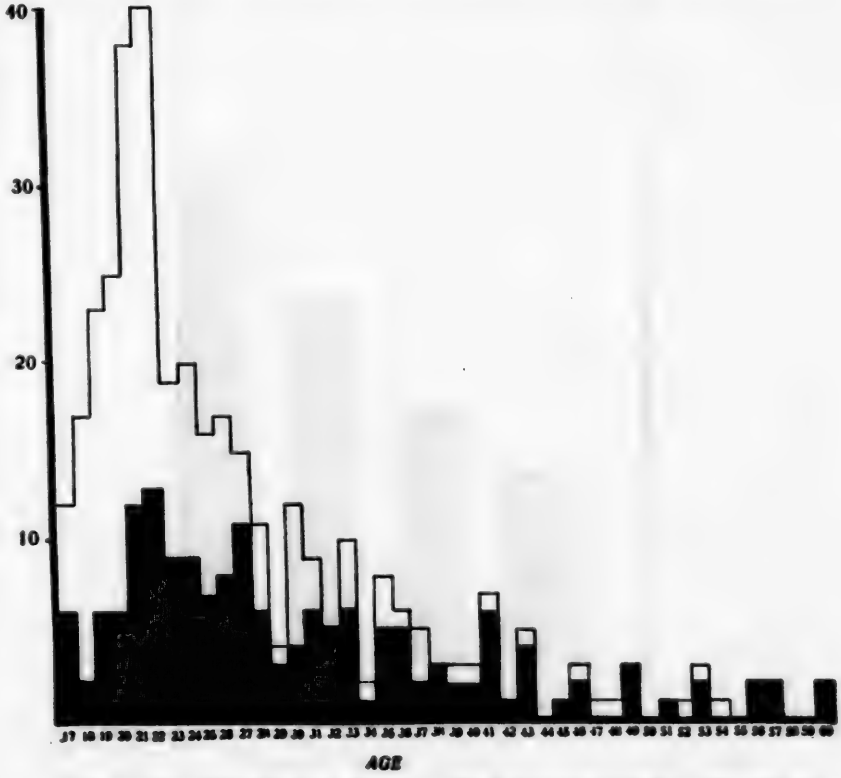

FIG. 2. The relation of age to infection with *Endamoeba gingivalis*. The height of each column indicates the number of persons studied in each age group. The age is indicated at the base of each column. The portion of each column in black indicates the number of individuals who possessed amoebic infection of the mouth.

days. This treatment was continued until two negative Wassermann tests indicated a cure. Throughout the treatment amoebas may be demonstrated in the gums in considerable numbers. Incidentally a number of these cases were ravaged with pyorrhea, although some

claim salvarsan to be specific treatment for pyorrhea, on the theory that spirochaetes are instrumental in causing the disease.

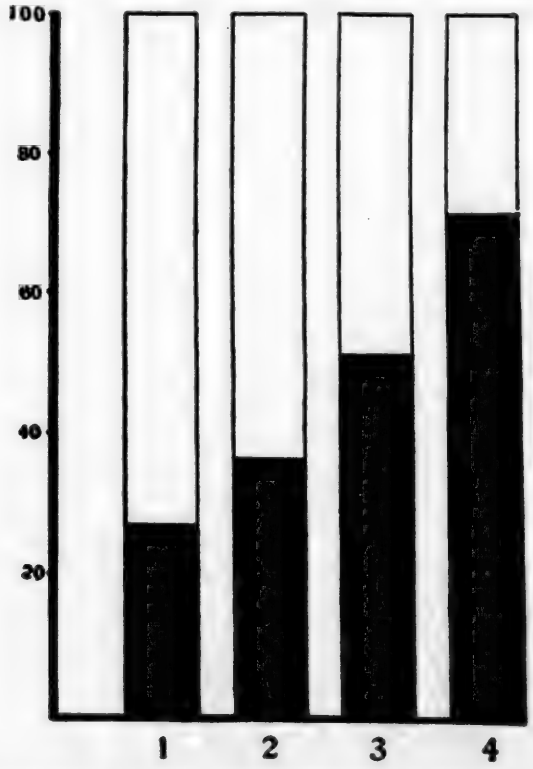

FIG. 3. The incidence of infection with *Endamoeba gingivalis* in each quartile of the age distribution curve of *fig. 2*. Column 1 represents an infection of 26.74 percent in the youngest one-fourth of all individuals studied. The incidence of infection is 36.03 percent, 51.05 percent, and 71.06 percent, respectively, in the successively older age groups (columns 2, 3 and 4).

We conclude that the amoeba can withstand concentrations of salvarsan and mercury salts, *in vivo*, that are lethal to the *Treponema pallidum*.

12. Relation to age

The ages of each of the 357 persons examined were obtained. They are plotted in *fig. 2*, wherein the total height of each column represents the number of individuals of the age corresponding to that column. The sections in solid black indicate those infected with *E. gingivalis*. It will be noted that we have examined young persons for the most part. The majority of those examined are between 18 and 26 years of age. In spite of the rather closely restricted age group dealt with, the increased incidence of infection with advancing age is striking. If the population as a whole had been dealt with, to include many above the age of 30 years, this increased liability to infection would have been much more striking. Because of the rather scattered distribution in the older age group, we have made comparisons of the incidence of infection in each of the quartiles of the curve. That is to say, four groups of equal size were segregated according to age, each containing 89.25 individuals. The youngest of these groups had an incidence of infection of 26.74 percent, the second 36.03 percent, the third 51.05 percent and the oldest, one fourth of all examined, had an incidence of infection of 71.06 percent. This is illustrated graphically in *fig. 3*.

We conclude from these data that, with advancing age, the probability of becoming infected with *E. gingivalis* increases at a rapid rate. The great majority of persons over 30 years of age (about 70 percent) are infected with this parasite.

13. Other correlations

Among 193 prisoners examined at San Quentin Prison were, first, those with undoubted advanced pyorrhea alveolaris; second, those with various pathological conditions of the mouth, doubtless including a number with true pyorrhea; and third, those whose mouths appeared relatively normal when rather superficially examined. The occurrence of parasites in 186 of these individuals has been published (Hinshaw 1926). The occurrence of *E. gingivalis* is shown in Table 11, where it may be seen that cases of advanced pyorrhea invariably harbor the parasite *Endamoeba gingivalis*.

A case of pyorrhea in the care of one of us (F. V. S.) is of interest in this connection—a young woman apparently in excellent health

afflicted with pyorrhea of unknown cause. We did not attempt a diagnosis of amoebas until the case had been under thorough treatment for a considerable period of time. The roots had been carefully scaled at frequent intervals for several weeks. "Viogen" and other disinfectants had been applied repeatedly. An autogenous vaccine had also been used. The diet had been so regulated as to provide every known vitamine, and a well balanced mineral content. In spite of all this the condition did not clear up, and a certain amount of pus continued to flow. There was some evidence that the process was spreading to other teeth than the lower incisors and cuspids, which were first involved. We made smears and cultures on several occasions, securing all the exudate available at any one time, but amoebas

TABLE 11

Occurrence of Endamoeba gingivalis in San Quentin Prison

	ADVANCED PYORRHEA	UNCERTAIN AND ANOMALOUS CASES	NORMAL MOUTHS AND INCIPIENT PYORRHEA
Number infected with *E. gingivalis*	51	51	18
Percentage of infection	100	77.27	23.68
Number free from amoebic infection	0	15	58
Percentage not infected	0	22.73	76.32

were never found. The smears were most extraordinary in one particular. Practically no microörganisms of any kind were present; but while polymorphonuclear leucocytes were fairly numerous, there were very few bacteria. In this remarkable case it was possible to count several polymorphonuclear cells to every coccus present, of which very few were isolated—and many less bacilli. There were no spirochaetes or fusiform bacilli, although these were carefully sought. Material removed from shallower pockets, and from between the teeth, showed many more bacteria, but the deep pockets were practically sterile. Our conclusion with regard to this singular case was that it arose from some obscure physiological malfunctioning; or possibly that the infectious agent had been removed by the dental treatment, but not before sufficient damage had been done to the tissues to prevent their immediate repair.

14. Summary of distribution

The incidence of infection with *Endamoeba gingivalis* in 357 individuals examined was 46.34 percent.

These parasites do not occur in the strictly normal mouth.

They are invariably found in typical inflammatory pyorrhea alveolaris, from an early stage of the disease until its termination.

Their presence is invariably associated with the existence and extension of gingival pockets.

They are more commonly found when the gum margin has receded (57 per cent).

Microscopic pus is always demonstrable in cases of amoebic infection of the mouth.

Hypertrophy of the gum may occur, but is not an invariable accompaniment of amoebic infection.

Supragingival calculus is usually present.

Subgingival calculus cannot always be demonstrated in early cases of amoebic infection, especially when the teeth are frequently scaled. In later stages of pyorrhea, it is always present.

Endamoeba gingivalis is less likely to occur in mouths with moderate amounts of detritus than in those with slight detritus, provided everything else is equal. This argues against the idea that it is a saprozoan feeding largely upon bacteria and food debris.

Conditions of the saliva associated with mild caries do not appreciably affect the amoeba of the mouth. Nor is there any demonstrable association between a history of caries and predisposition to amoebic infection.

The inflammatory reactions associated with amoebic infection are in the nature of a chronic process. *Certain acute localized inflammatory processes do not support E. gingivalis.*

Persistent and intelligent efforts of the patient to secure good mouth hygiene are not effective in destroying this parasite.

The saliva of amoebic hosts is usually either plentiful or of a consistency that forms a glairy film on mucous surfaces.

Endamoeba gingivalis thrives in a concentration of neosalvarsan and mercury that is lethal to *Treponema pallidum.*

The incidence of infection with the amoeba of the mouth is very

much higher in persons belonging to older age groups. The incidence of infection was found to be 26.74 percent, 36.03 percent, 51.05 percent, and 71.06 percent, respectively, in the four quartile groups arranged from youngest to oldest.

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THE INFECTED VITAL DENTAL PULP[1]

An Important Focus of Systemic Disease

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I. INTRODUCTION

Research and clinical observations, carried on for the last two years, have disclosed that chronic infection of the pulp in so-called vital teeth is a particularly potent focus of infection. It is most frequently overlooked in oral examinations because it is very difficult to diagnose.

The term "vital pulp" in the minds of a great many is synonymous with normal, healthy pulp. Vitality, however, refers only to the functioning of physiological processes. If they function, an organ is alive; if they do not function, it is dead (necrosis). As long as metabolism takes place we may speak of the pulp as vital, but at the same time it may be diseased, just as a person may be alive and at the same time sick. Another paradox is presented by the so-called testing of the teeth for vitality. Many clinicians believe that a tooth reacting positively to various applications of the electric current is

[1] Read at a session of the Sixth Annual Meeting of the International Association for Dental Research, Washington, D. C., March 25–28, 1928. An abstract was published in the last preceding issue: *Journal of Dental Research*, 1928, viii, p. 412. The paper was also presented, on February 10, 1928, at a meeting of the Boston Section of the International Association for Dental Research.

"alive" or normal. Such tests, however, afford proof of electric conduction, but not of normality. They do not determine whether the metabolism of the pulp is functioning, nor are they an indication as to whether the organ is healthy or diseased. A patient may jump when receiving an electric shock, but this does not prove that he is not ill. The dental pulp, therefore, may be (*a*) vital and in normal health; (*b*) vital and diseased; or (*c*) non-vital or dead (necrotic).

The special pulp affection with which we are concerned in this paper is chronic infection of the vital pulp; that is, chronic infection of a pulp in which there is circulation and nerve response is undisturbed. The condition may continue for several weeks or months, which is contrary to the general idea. Infection of a pulp, through consequent hyperemia of the dentinal artery in the apical foramen, was believed to cause strangulation of the dentinal veins and stasis of the circulation, leading in a short time to pulp necrosis. This investigation has proven that an infected pulp may remain vital for a long time, and meanwhile be a focus from which continuous absorption of bacteria and their toxins may take place.

II. ETIOLOGY

The infection is almost always the result of dental caries. The type of caries may be primary, but generally it is secondary and occurs under fillings, gold crowns, porcelain jacket crowns, or bridges. It may be new caries starting at the margin of a filling, or a continuation of bacterial activity in a poorly cleaned or imperfectly sterilized cavity. In rarer instances, the pulp infection is derived from extension through a pus pocket at the side of the root of the tooth, or from an infection in a neighboring tooth. (See *figs. 11–16.*)

III. BACTERIOLOGICAL EXAMINATION

To determine the bacteriologic condition of an infected vital pulp, the same technique was followed as is employed for pulpless teeth: the gum was painted with tincture of iodine and a piece of sterile gauze placed on each side of the tooth. After extraction, the apex of the tooth was cut off with sterile cutting forceps in such a manner that the apex jumped directly into the culture tube held ready by an assistant. The culture tube contained Locke's solution with gravel

FIG. 1. Section through an incisor tooth showing pulp in state of hyperemia.

FIG. 2. Longitudinal section through maxillary incisor, showing general chronic pulpitis.

A.—Adventitious dentine due to caries. *B*.—Adventitious dentine deposited in response to erosion. *C*.—Infected adventitious dentine.

and a trace of gelatin. In this manner, the bacteria are collected from the apical part of the root canal, where there is less chance for contamination than in the crown of the tooth near the cavity. Later the method was improved. At present, as well as in some of the specimens mentioned in *Table 1*, the apex of the tooth, after extraction, is washed on the outside with alcohol so that the culture must come from the content of the root canal of the apex. When the

FIG. 3. Longitudinal section through molar tooth. Ulcerating chronic pulpitis in the left cornea of the pulp.

specimen is in the culture tube, it is necessary to shake well in order to cause maceration.

The twenty-five (25) cases of infected vital pulps summarized in *Table 1* were secured as specimens in the manner just described, and then cultured by Dr. Calvin G. Page, whose great interest and painstaking investigation is gratefully acknowledged, although this paper includes only a preliminary report of his part of the work. The teeth with pulp infection were mostly selected from patients having systemic diseases that could reasonably be ascribed to a dental focus. The largest number were cases of arthritis; others included eye, ear, or kidney infections, also phlebitis and toxemia.

TABLE 1

Bacteriological examination of cases of chronic pulpitis

TYPE OF DENTAL CONDITION	CASES		PATIENT	TOOTH	LOCAL SYMPTOMS	SYSTEMIC CONDITIONS	PRELIMINARY BACTERIOLOGICAL REPORT
	Number	Total					
Pulp chamber closed with large filling in crown	1		Mr. A. P.	R. U. Bic.	None	Lumbago	Strep. viridans
	2		Mr. L. M. G.	L. U. Cusp.	Swelling on face	None	Strep. gram +; acid forming
	3		Mr. F. S.	L. L. 2 Mol.	None	Arthritis	Strep. gram +: probably strep. viridans
	4		Mrs. C. B.	L. L. 2 Bic.	None	Phlebitis	Strep. viridans
	5		Miss M. B.	L. L. 1 Mol.	None	Retrobulb. neuritis	Strep. viridans
	6		Mr. R. S.	L. L. 1 Mol.	None	None	Strep. viridans; also non-hemol. strep. and bacillus
	7		Mrs. Ch. D.	L. L. 1 Mol.	None	Arthritis	Strep. viridans
	8	8	Mrs. M. S.	R. L. 1 Mol.	None	Toxemia, pallor	Strep. gram +; viridans
Same, with gold crown	9	1	Dr. E. Y.	L. L. 2 Mol.	None	Arthritis	Strep. viridans; also gram − staph.

Same, with deep unfilled cavity but no perforation	10		Miss M. T. D.	L. L. 2 Mol.	None	Nephritis	Strep. gram +; distinct hemolysis
	11		Mr. L. S.	L. L. 1 Mol.	Pain	None	Strep. gram +; slight hemolysis; prob. str. viridans
	12		Mr. L. H.	R. L. 2 Mol.	None	Deafness; 3 weeks	Streptococcus
	13		Mrs. J. C.	L. L. 1 Bic.	None	Neuralgia	Strep. viridans
	14		Mrs. A. J. A.	R. L. 1 Mol.	Sensitive to percussion	Arthritis	Strep. viridans
	15		Miss S. L. F.	L. L. 1 Mol.	None	Arthritis	Strep. viridans
	16		Mr. O. A. H.	R. U. Cusp.	None	Infect. in nose	Strep. viridans; and yellowish staph.
	17		Mr. J. H. S.	R. U. Cusp.	None	Toxemia; low basal metab.	Streptococci
	18	9	Dr. G. C.	R. U. 2 Bic.	None	Antrum infect.	Diplococci and white staph.
Infection caused by neighboring tooth	19	1	Mrs. M. J. Mc. D.	R. L. Lat. I.	Acute abscess	High bl. pr.; dizzy	Strep. viridans
Infection caused by pocket on side	20		Mr. C. H. C.	L. L. 3 Mol.	None	Rheumatic fever	Bacillus gram −; and gram + in chains; diplococcus gram −
	21	2	Mrs. S. L.	R. L. 3 Mol.	None	Arthritis	Strep. viridans
Pulp chamber open, with perforation from cavity	22		Mrs. J. C.	R. L. Cusp.	None	Neuralgia	Motile bacillus
	23		Mr. W. A. G.	R. L. 1 Mol.	None	Arthritis	Streptococcus not isolated —spore bearing bacillus and large coccus
	24		Mr. J. W. A.	R. L. 3 Mol.	Pain R. L. side	None	Strep. and staph.
	25	4	Miss D. E. G.	L. U. 3 Mol.	Pain L. U. side	Arthritis	Strep. gram +; gram + bacilli

Table 2 shows that the Streptococcus viridans is the most important factor in chronic pulp infection. From sixteen out of the twenty-one cases, with pulp chambers closed, pure cultures of streptococci were produced. Cases with an opening into the pulp canal from the cavity, or with a deep pus pocket on the side of the root, showed mostly mixed infections.

IV. PATHOLOGICAL DEVELOPMENT

The progress of the infection, from dental caries, is made by an advance of the bacteria in the dentinal canaliculi. The resultant toxins are taken up by the circulation in these canaliculi and so

TABLE 2

Classification of bacteriological findings

TYPE OF DENTAL CONDITION	ORIGIN OF PULP INFECTION	TOTAL NUMBER OF CASES	DATA ON DISTRIBUTION: NO. OF CASES			
			Streptococci alone	Streptococci and other cocci	Streptococci and bacilli	No streptococci; other mixed infections
Pulp chamber closed	Caries, filling, crown	18	14	2	1	1
Pulp chamber closed	From neighboring tooth	1	1	—	—	—
Pulp chamber closed	From pocket on side	2	1	—	—	1
Pulp chamber open	From perforation due to caries	4	—	1	2	1
All conditions	From any of the causes	25	16	3	3	3

reach the odontoblastic layer of the pulp. These toxins are not strong enough to destroy the cells, but are sufficiently irritating to stimulate the odontoblasts to new activity. This then results in the formation of adventitious dentine in the region where the injury occurs. In a way this is a protective reaction, but unfortunately the newly formed dentine furnishes only a temporary barrier, and the infection progresses through the new dentine until it finally reaches the pulp. Owing to the type of injurious agent (Streptococcus viridans) and the circumstances under which the injury occurs, the nature of the reaction in the tissue is that of chronic infection. This is a slow, gradually progressive and long continued process, which, however, may change its characteristics. It is so gradual in onset that it fails to produce the circulatory changes of acute pulpitis, which results

generally in strangulation of the circulation and complete pulp necrosis. The circulatory phenomena in chronic pulpitis are rarely interrupted. Nor is there a suppurative process active enough to destroy the nerve fibres. The pulp and tooth, therefore, retain their blood circulation and their sensation, as stated in the beginning of

FIG. 4. Longitudinal section through molar. Extensive chronic ulcerating pulpitis.

this paper. This condition may continue for an indefinite time and, through hematogenous absorption, act as a focus of infection.

If secondary infection occurs, it results in an entire change of the pathological picture. Pyogenic organisms may produce suppuration with all the symptoms of acute infection. Saprophytic bacteria produce gangrene of the pulp, and sometimes there is gas formation (Bacillus aerogenes capsulatus).

Effect on surrounding tissue. Chronic pulpitis soon causes a reaction in the apical part of the peridental membrane, which is of greatest importance in diagnosis, as will be seen later. This periapical inflammation is slow in its onset and mild in nature, and generally causes no special symptoms. Round-cell infiltration occurs, which gradually produces a swelling of the peridental membrane. This in turn results in the enlargement of the space between the tooth

FIG. 5. Longitudinal section through bicuspid, with perforation from the cavity into the pulp chamber. Chronic ulcerative pulpitis discharging through the exposure.

apex and the alveolar socket owing to absorption of the lamina dura. Proliferating periodontitis and the formation of a so-called dental granuloma may be the final result.

V. HISTOLOGICAL PATHOLOGY

In the microscopic study of the earliest stages of pulp involvement, we find infection of the canaliculi of the primary dentine and of adventitious dentine if present. Marked hyperemia occurs in the

FIG. 6. Longitudinal section through mandibular first molar of a child. Chronic hypertrophied pulpitis with perforation of the pulp chamber.

A.—Cavity. *B*.—Hypertrophied pulp tissue. *C*.—Periapical reaction at the apex. *D*.—Large apical foramen.

pulp (*fig. 1*). Mononuclear cells invade the tissue very soon (*fig. 2*). Eosinophiles, lymphocytes, endothelial leucocytes, and plasma cells, are present, the latter in greatest number. In cases of longer duration, we may see either a diffuse chronic inflammation, with a general

FIG. 7. Longitudinal section through molar showing thick layer of infected dentine. *aa*. Infected dentinal canaliculi. *B*. Chronic pulpitis, with focal abscess.

infiltration by these cells (*fig. 2*), or focal accumulation in one or several places in the pulp (*fig. 7*).

Ulcerating chronic pulpitis. The cases with diffuse inflammation may later cause dissolution of tissue in the region where the injury

FIG. 8. Longitudinal section through mandibular molar. Chronic pulpitis, with advanced abscess formation.

A.—Abscess, with necrosis in centre. *B*.—Abscess, with pus discharging through perforation into cavity. *C*.—Reaction in peridental membrane.

started. This often occurs in the cornea of the pulp (*fig. 3*). Sometimes a large part of the pulp may become necrotic, forming a large ulcerating surface (*fig. 4*). If there is a perforation into the carious part of the tooth, it gives opportunity for discharge from the ulcer (*fig. 5*).

FIG. 9. Longitudinal section through mandibular third molar. Symptoms of severe pain.

A.—Abscess of sub-acute type, with polymorphonuclear leucocytes predominant. *B.*—Cavity. *D.—Dentine. N.*—Nerve fibres pressed to the side by abscess. *P.*—Pulp in state of hyperemia.

Hypertrophic chronic pulpitis. In teeth of young patients we often find a proliferation of the pulp tissue. The stimulation from the toxins of the infection causes the formation of new tissue, made up of capillaries, fibroblasts, and plasma cells. It grows through the aperture into the cavity of the tooth, and often becomes covered with epithelium (*fig. 6*).

Chronic pulp abscess. In the other cases, small abscesses form in

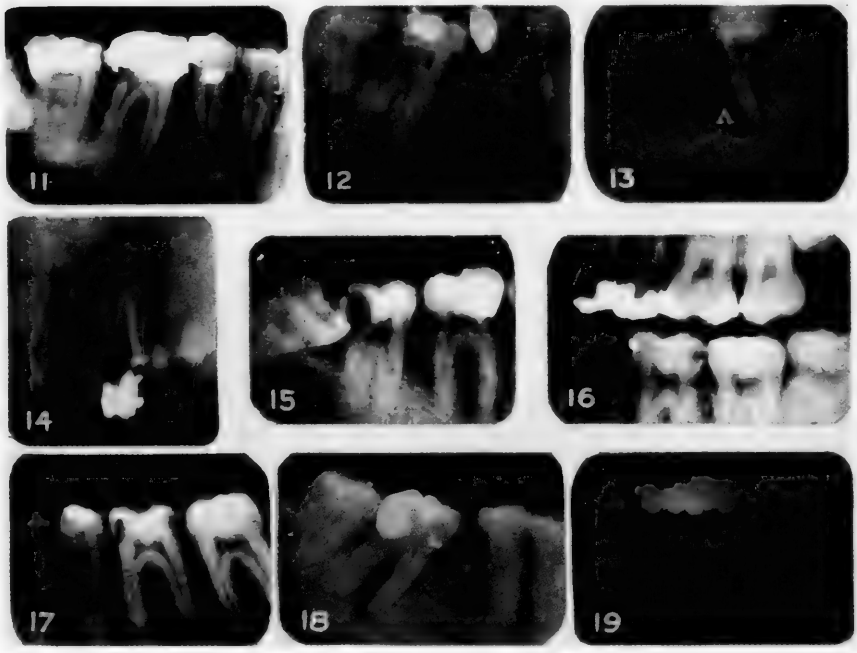
the various parts of the pulp (*fig. 7*). Polymorphonuclear leukocytes are attracted and suppuration occurs. The pus at first is absorbed, but later, when an opening has formed an outlet into the pulp chamber, drainage into the mouth may be established (*fig. 8*). Such pulp

FIG. 10. Higher magnification of abscess shown in Fig. 9

abscesses may at times show a picture made up of a large accumulation of polymorphonuclear leukocytes (*figs. 9* and *10*). In all these cases, even if the abscessed area is large and contains a great deal of necrotic tissue, the abscesses are well circumscribed, and the blood vessels and nerves of the pulp are not destroyed.

VI. SYMPTOMS

Subjective: local. In most cases there are no subjective symptoms at all. But occasionally a patient can remember that at one time or another he felt a sharp pain of short duration, but could not localize it. A dull throbbing is felt in exceptional cases, which is usually



FIGS. 11–19. Roentgen pictures showing indirect evidence of pulp infection

noticed when exercising or lying down. Some patients also complain of a dull pain from hot food, which is due to either a supersensitive condition of the nerves, or the presence of gases in the pulp chamber as seen in gangrenous pulpitis.

Reflex. Reflex symptoms occur quite frequently in chronic pulpitis, but they are by no means typical of the disease. The pain is probably caused by a toxic irritation of the nerve fibres in the pulp, and usually

FIGS. 20–27. Roentgen pictures illustrating the case reports

is very obscure and referred to any part supplied by the trigeminal nerve on the same side of the face (neuralgia dentalis). It may also be referred to other nerves of the face through anastomosis, especially the tympanic plexus of the ear (otalgia dentalis).

General. General symptoms are very frequent. A slightly raised temperature, from about 99° to 100°F., is not unusual, especially in the afternoon. More frequently we find cases without raised temperature, but presenting the various conditions associated with focal infection, especially vague medical symptoms, such as fatigue disproportionate to the slightest exertion; inability to do the accustomed day's work, mental or physical; the requirement of an abnormal amount of sleep; and greyish, sallow skin. Very common are pains in the muscles, swelling or pain in the joints, or neuritis in any part of the body. Other organs may become infected, such as the eye, the heart, or the kidneys.

VII. DIAGNOSIS

In the absence of subjective local symptoms, the patient is not aware of chronic pulp infection. And in cases of general disease, when routine examination of the teeth is made, the diagnostician must pay special attention to the condition of the dental pulps. An examination should consist of a careful roentgen study, but the findings should be checked up by clinical examination.

Roentgen examination. In addition to a routine set of roentgen pictures, it is often necessary to study individual teeth by means of further exposures made from various angles. It goes without saying that, with special experience, the diagnostician will improve his technique as well as become more and more proficient in the interpretation of the findings.

Direct evidence of pulp disease. Infection of the pulp cannot be directly demonstrated because the ensuing changes are histological in nature and do not affect the radiability of the tissue. The deposit of adventitious dentine in the pulp canal can be recognized by the consequent decrease in size. This may be seen as an obliteration of the coronal part of the pulp in single-rooted teeth, or of the cornea of the pulp in multi-rooted teeth.

Indirect evidence of pulp infection. Indirect evidence presents

itself in the roentgen picture (a) as an etiological factor; (b) as an extension of the inflammation into the periapical area.

a. The finding of an etiological factor directs attention to a tooth that often should be further investigated either by means of additional roentgen pictures or by clinical methods. Etiological factors are seen as small defects representing caries under fillings and crowns, and extending close to the pulp canal (*figs. 11* and *12*). In rarer instances deep pockets may be seen on the sides of teeth (*fig. 13, A*), or the infection on a neighboring tooth may extend to the apex of a tooth with a normal pulp (*fig. 14*). Sometimes an impacted third molar may cause decay in the distal side of the second molar, producing pulp infection (*fig. 15*). Defects in the crowns of teeth are not always easily demonstrated in the ordinary roentgen film, and it may be necessary to use the bite-wing method (*fig. 16*).

b. The secondary changes caused in the periapical tissue are evidence, almost *per se*, of pulp infection. In the beginning the enlargement of the periapical space, produced by the increased size of the inflamed peridental membrane, is so slight that it is visible only in a roentgen picture of good quality and taken at a favorable angle (*fig. 17*). A positive diagnosis can often be made only when roentgen pictures also demonstrate the cause of the condition, or when there is confirmation by clinical examination. In cases of longer standing, the periapical evidence is well marked and unmistakable. (*Figs. 18* and *19*).

Clinical examination. In taking the history, the questions should be directed so as to bring back to the patient's mind half-forgotten minor disturbances. Among these are sharp pains of temporary duration, or dental neuralgia of more or less lasting character. (Case of Dr. F. P. D., below.) Pain may have been caused by hot food. Specially painful experiences during past dental operations may be remembered. Sometimes a patient knows that a cavity was extremely deep, or that the pulp had almost been exposed. The dentist may have said he did not dare to remove all the decay, or the patient on request may remember that a tooth had been aching after it was filled, but the pain had later disappeared. Such information may often be found valuable, and in conjunction with the x-ray findings lead to a definite diagnosis.

As far as clinical tests are concerned I have found that the heat test is practically the only one of any help. In chronic pulpitis the nerves of the pulps seem to be particularly sensitive to heat. In some cases, where there is gas formation, the heat causes expansion and pressure on the nerve endings. To make the test we use an instrument to which is attached a ball of gutta-percha. This is heated and applied to the tooth. If pain is experienced, especially the kind that has the tendency to be sustained, it is a pretty good indication of pulp disease. Electric tests are of no great diagnostic value. With them we may distinguish between the root-filled and the vital tooth, but the variations of response in infected vital pulps are insignificant and offset by changes in conductivity caused by large fillings or deposits of secondary dentine.

VIII. CASE REPORTS

Mr. D. R. S. This is a case of neuralgia in the left side of the face. The roentgen picture shows an unerupted lower third molar. The patient was advised to have it extracted to relieve the symptoms. A new roentgen picture, however, revealed in addition an area under a filling of the second molar indicating caries. In addition, there are very slight changes in the apices of the tooth, and the two findings permit a positive diagnosis of pulp disease in this tooth. The second film shows the same tooth after the filling had been removed. (*Figs. 20, a and 20, b.*)

Dr. F. P. D. The patient has had symptoms of general toxemia with lameness of elbows, hands, wrists, and knees. Roentgen examination disclosed a large cavity in the distal side of the right upper second molar. He had felt a sharp pain in his tooth about one week ago. Diagnosis of pulp infection was made and the patient was advised to have the tooth extracted. He thought the matter over for two days and finally decided to submit to the treatment. The extraction of the tooth promptly relieved all the symptoms. (*Fig. 21.*)

Miss B. P. A young college student came to me in January, 1916, complaining she felt all tired out, and every evening had a rise in temperature to about 99.5°F. She had been suffering with this condition for several months without improvement. Tuberculosis was suspected. All her teeth were x-rayed, the pictures revealing changes at the apex of the left lower first molar and bicuspid. The root canals of the molar showed no signs of treatment or filling. On examination the patient stated that she had never suffered from any pain in this tooth. A note was made in

the record that the tooth was vital when drilled into. After the tooth was extracted, the temperature remained normal and the patient's general health improved rapidly. (*Fig. 22.*)

Mr. R. O. He was seen first in September, 1923, having suffered from arthritis for six months. It started in the right knee, then went to the left knee, leg, and foot; later to the arms and hands. He had a great deal of pain and swelling in the joints. He was confined to bed. Roentgen examination of the teeth showed changes in the periapical tissue of both the right upper second molar and the right lower second molar, with pulp infection. In the upper tooth this was caused by a deep cavity; in the lower one, by a capped pulp. In addition the left upper first and second bicuspids showed root-canal filling, and slight changes at the apex indicating infection. The four teeth were extracted and the patient recovered completely from this multiple arthritis, and now reports (January 9, 1928) that he has not had the slightest recurrence of the disease. (*Figs. 23, a, 23, b, and 23, c.*)

Miss Mc. I. In April, 1925, the patient complained of swelling in both knees and arms, with intense pain. Routine examination of the teeth disclosed a large periapical disturbance in the right lower central incisor, apparently due to pulp infection in this tooth. The tooth was extracted in April, 1925. The patient reports (January 8, 1928) that she was able to walk one month after the extraction, and has been well ever since. (*Fig. 24.*)

Miss M. B. On October 10, 1927, the patient, a school teacher, had been suffering for five days from retrobulbar neuritis. Examination of the teeth revealed a certain amount of pyorrhea. Roentgen pictures disclosed a filling extending near the pulp of the left lower first molar. The tooth was extracted on October 10, 1927. A culture was made from the apical part of this tooth, the bacteriologist reporting infection by Streptococcus viridans (pure culture obtained). On November 2, the patient had better vision and was improved. On December 5, with sight nearly normal, the patient had gone back to work. January 7, 1928: the patient reports she has been entirely well during the intervening month. (*Fig. 25.*)

Mrs. L. F. W. This is a case of pregnancy. On August 25, about the sixth month, the obstetrician found a blood pressure of 146; urine normal. He discovered that for several weeks the patient had had trouble with a tooth. The roentgen picture of this tooth showed extensive changes around the apex, apparently caused by chronic pulp infection. The tooth was extracted by a colleague. On the next visit, September 26, the blood pressure had dropped to 122. (*Fig. 26.*)

Dr. E. Y. The patient had had pain and swelling in the right wrist,

which caused discomfort when using the hand. Examination showed roentgen evidence of periapical changes on the left lower third molar, indicating chronic pulpitis. The tooth was extracted and a culture made from the apical part. The bacteriologist reported a slightly hemolytic streptococcus and a gram-negative staphylococcus. Exacerbation of the symptoms occurred the next day, but disappeared entirely on the second day. In a few days the wrist was entirely well. (*Fig. 27.*)

IX. CONCLUSIONS

(1) All teeth that have been painful, or very sensitive to hot food, should be carefully investigated, because such teeth often have infected vital pulps.

(2) The fact that a tooth reacts positively to so-called vitality tests, or is sensitive when opened up, or painful when the pulp is exposed, does not prove that the pulp is not infected.

(3) A more careful study of the roentgen picture is necessary. If a large number of root-filled teeth, with or without infection, are present, they often distract attention from the small changes giving indirect roentgen evidence of an infected vital pulp.

(4) In addition to the roentgen examination, a clinical examination of all suspected teeth should be made.

(5) The use of a dental diagnostician should be encouraged. He would act as a consultant with whom the various aspects of the case could be discussed. The general practitioner should find it of great advantage to employ the services of one of our well-trained specialists rather than to send his patients to a commercial x-ray laboratory, where men with questionable professional qualification make business of one of the most important branches of dentistry.

(6) An infected vital pulp, no matter whether there are well marked periapical changes or not, is often a very important focus of infection. Its removal gives promise of prompt relief of the systemic condition.

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